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Acute Carbon Monoxide Poisoning

Its Diagnosis After Death

In the previous issue of this *Journal,* Simpson[1] made a distinguished and fundamental contribution to the important subject of acute carbon monoxide poisoning. His survey emphasized the importance of assessing the amount of carbon monoxide in the blood in relation to the clinical and necropsy findings. While there are high concentrations of this poison in the blood which are clearly inconsistent with life, there is a considerable lower range which may not be fatal in normal persons, but which may account adequately for death in e.g. "the decrepit, the diseased, the drugged and the drunk", the 4 D's of carbon monoxide poisoning.

Simpson has also established convincingly the valuable practical point that in a dead body—short of dissolution by putrefaction—a delay of even days is of little moment since there is no evidence for the mythological view that rapid disappearance of the gas occurs *post mortem.* Indeed, the evidence is reliably the other way, even in respect of blood in the test tube.

Death from carbon monoxide poisoning presents an interesting problem for differential diagnosis, readily settled by an analysis of the suspected blood. The striking, bright, cherry-red colour of the skin, the blood and the internal organs of many victims should raise immediate suspicion about this cause of death. Yet Simpson's data indicate that 46% of his cases were unrecognized before necropsy; and in communities containing dark or black-skinned populations, the problem must be even greater. Moreover, there are for exclusion other conditions which can produce a bright red colour in the dead.

1. Simpson, K. (1955): This *Journal,* 2, 5.

It has long been recognized that in death from exposure to cold the victim may be of a bright, arterial hue.[2-6] Uttley[7] (who has made a special study of the post-mortem appearances in these cases) describes this bright red colour of the blood as characteristic of deaths from cold. Some writers have attributed the colour of the blood to an entry of oxygen through the frozen skin to form brightly coloured oxyhaemoglobin in the superficial blood vessels after death.[3] It is doubtful if this is the correct explanation, as the internal blood also has this colour. Moreover, we have occasionally seen the development of this appearance in bodies placed for storage in refrigerators. The phenomenon has also been observed in the living, e.g. ice packs (placed around the feet to reduce the metabolic requirements of the tissues in a case of ergot poisoning with severe spasm of the peripheral blood vessels) have produced such a bright red appearance of the localized parts being cooled, that at first there was an assumption (proved entirely wrong by subsequent events) that the patient's clinical condition was improving.

The nature of this frigid pigment which can occur in the living and the dead on exposure to cold is well worth spectroscopic investigation, *inter alia*.

The appearances of death from carbon monoxide can equally easily be simulated by cyanide poisoning, where the tissues are deprived of the power to remove oxygen from the arterial blood passing through them. Consequently the arterial blood passes into venous circulation largely unaltered, producing an appearance which may need to be distinguished from carbon monoxide poisoning.

As nitrites can also produce in the blood, after death, a compound with a reddish colour resembling carboxyhaemoglobin,[8] it is clear that those who die 'in the pink' provide an interesting problem which may need laboratory aid for its solution.

2. Smith, S. and Cook, W. G. H., ed. by (1948): *Taylor's Principles and Practice of Medical Jurispondence*, 10th ed., **1**, 483. London: J. and A. Churchill, Ltd.
3. Strassmann, G. (1954): In *Legal Medicine*, ed. by R. B. H. Gradwohl, p. 198. St. Louis: C. V. Mosby Co.
4. Smith, S. and Fiddes, F. S. (1949): *Forensic Medicine*, 9th ed. p. 247. London: J. and A. Churchill, Ltd.
5. Kerr, D. J. A. (1954): *Forensic Medicine*, 5th ed., p. 147. London: A. and C. Black.
6. Glaister, J. (1950): *Medical Jurisprudence and Toxicology*, 9th. ed., pp. 170 (Fig. 48), 232.
7. Uttley, K. F. M. (1948): New Z. Med. J., **47**, 427.
8. Strassmann, G. (1954): *Op. cit.*, p. 288.

The Expert Witness—A New Look‡

Rowland H. Long*

I accepted the invitation of your Academy because I had something I wanted to say, and this seemed to me a most appropriate place for saying it. My remarks will be in no wise sensational but rather of the commonplace, so I shall give them in the most direct fashion of which I am capable. I speak to you as a trial lawyer who has tried between 750-1,000 cases involving medico-legal questions in the last 25 years. All too much has been decried about the untrustworthiness of physicians as expert witnesses. I have found that physicians by and large are impartial, truthful men, many of whom have expressed candid scientific opinions on the witness stand, the ululations of disappointed litigants and academicians to the contrary. I am well aware that a facile conscience may stretch itself like india rubber when a physician is testifying as a biased, partisan witness, or when his fee is contingent on the outcome of the case. There is a small coterie of physicians who do not come to court to inform the jury but come as advocates determined to color testimony, which lies almost wholly in their control, to the requirements of their employers. There is a problem presented in all cases requiring expert medical testimony to aid the jury in the solution of certain issues. The problem is twofold: What constitutes an expert, and how will expertness be determined?

In 1899 the Court of Appeals of New York asserted that 'If any man be in reality an expert upon any given subject belonging to the domain of medicine, his opinion may be received by the court.'[1] This would be a fair work-a-day rule if there was not an unfortunate tendency on the part of the courts to permit self-styled medical experts to usurp the witness stand. An illustration will present my conception of what does not constitute a medical expert.

A specialist of high repute was sued for damages for causing loss of sight due to the alleged negligent performance of an ethmoidal operation. The plaintiff produced a 26-year-old physician who had been licensed to

‡ An address delivered at the Annual Meeting of the American Academy of Forensic Sciences in 1954.

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practise medicine for 3 years, including an internship of 18 months. This fellow was not a member of any hospital staff and had never performed an ethmoidal operation. The New York Supreme Court, however, admitted his opinion that defendant did not operate in accordance with accepted surgical procedure. His cross-examination reads in part thus:[2]

Q. Before you go in, as you do here as an expert, you first try to find out what the plaintiff is trying to prove, don't you? A. Yes.

Q. And then you see how much help you can give him, don't you? A. Yes.

Q. And how much can you support what his contention is? A. Yes, sir.

Q. And that is part of the business in the deal, isn't it? A. Part of my work is expert testimony.

Q. You never performed a major operation on the abdomen, have you? A. Not myself.

Q. But you would assume to undertake, wouldn't you, to-morrow morning, to go into court before a jury and testify as an expert as to how an abdominal operation should be performed? A. If the case were just, I would.

Q. You have never operated on the brain, have you? A. No, sir.

Q. You would go into court and before a jury and testify as an expert on brain surgery, wouldn't you, if you were hired? A. If the case were just.

Q. And if you were hired? A. If the case were just and I were hired, I would.

This case does not stand alone. Scores of so-called medical experts throughout the United States are permitted by trial courts to give testimony concerning specialties in which they have neither special training nor experience. Trial courts have inherent power to set satisfactory standards of expertness and have power to exclude an unqualified physician as a witness. There has been a hue and cry by academicians for legislation to change the rules of evidence relating to expert opinion evidence. I do not know of any demand among trial lawyers who bear the brunt of presenting expert testimony to juries for any sweeping revision of the rules of evidence. On the contrary, I think it is clear that such schemes were conceived and fostered by academicians and some others without adequate consideration of the important factors involved or the present power inherent in the courts to make corrections without any legislation.

Among the law professors who urge a change in the rules is Professor Wigmore, who has this to say:[3] 'As soon as a clash of opinion appeared between experts paid by the parties, the jury would turn for its *real* guidance to the impartial one selected and summoned by the judge and his conclusion would have an almost decisive weight with the jury.' Impartiality and competency are not synonymous. I am acquainted with the personal physicians of some judges, and I would not trust some of them to pare a corn. A judge may be impeccable but he is not infallible, and his selection of an expert does not spell competency. But Professor Wigmore would have the case turn on the evidence of the judge-selected expert who might disagree with all others. Wigmore continues: 'At the same time there would be no risk of suppressing *possible* truth by excluding the parties' experts.' This sentence carries the ugly insinuation that difference of

opinion is bottomed on lack of truth. Is *possible* truth only when the experts of the parties agree in their opinions? 'And', says Wigmore, 'the experts called by the parties would in fact be of service in exposing occasional errors of the *official* expert.' Does this suggest a third opinion differing in all or some respects from the others? Wigmore has told us that opinions of the 'official expert' would have almost decisive weight with the jury, and I am sure he is right, so they would give decisive weight to the errors as well.

Professor Wigmore suggested three possible remedies to break down the established rules of evidence dealing with expert opinion evidence:

1. An official jury of experts where a scientific fact is at issue. He concedes that this suggestion would be unconstitutional, because the scientific fact is usually only one issue in the case.

2. The use of only official experts as witnesses. He rejects this suggestion because he feels that it would commit certain issues to a body of men who, under certain local and political conditions, would be unreliable.

3. An expert selected by the trial judge. He asserts that this proposal would eliminate most of the abuses of the present system and that it need only be used when there is a clash of opinion between experts of the parties.

Since unanimity of opinion among experts is not to be expected, the judge-selected expert would dominate the case. Domination of a jury by anyone should be avoided.

In 1937 the National Conference of Commissioners on Uniform State Laws submitted for consideration and adoption by the states a model bill entitled *Uniform Expert Testimony Act*. So far as I know, it has not been enacted in its entirety in any state. Its enactment would not eliminate the defects which it purports to do for these reasons:

1. It does not provide standards for determining expertness.

2. It does not eliminate the personal equation in the selection of experts either by the court or litigants.

3. It does not eliminate the evil associated with the contingent fee.

Other suggestions for remedial legislation have been made by lawyers and physicians.[4]

I feel that the solution of the problem does not require legislation. It does not require the abandonment by litigants of the right to select their own experts. It does require alert trial judges whose duty it is to preclude untutored pretenders and charlatans from the witness stand.

Forty odd years ago a committee of the New York State Bar Association submitted a report in which they set forth the evils prevailing in medical expert testimony. It reads, in part, as follows:

Want of satisfactory standards of expertness with its result of inviting the testimony of charlatans . . .

An unfortunate tendency upon the part of some trial judges to permit incompetent so-called medical experts to testify to opinions predicated upon widely unrelated facts and under oath to express views which are but the speculative vagaries of ill-informed minds.

Remedies have been suggested. None in my opinion will work so well as the present method of permitting each party to adduce such testimony

as he may consider essential to establish the theory of his case. If this useful method of presenting proof is to survive, courage must be employed unstintingly by trial judges; and trial lawyers and medical experts must maintain a nice sense of allegiance to the standards of their professions.

The trial judge should not hesitate to hold that a witness is incompetent to testify, whenever the facts fairly show that the physician is not qualified by study, training or experience in the medical or surgical specialty which is the subject of investigation. As long as trial judges refuse to recognize that a licence to practise medicine is only a facet of the qualifications of a witness, it will usually be possible to find some charlatan whose theories will permit him to testify as his employer wishes even though no experienced specialist would agree with him. A trial judge should abandon the idea that a jury will unerringly distinguish the competent expert from the pretender or charlatan.

Definitive criteria in guidance of the trial judge's determination of the qualifications of an expert witness rest primarily on 'occupational experience' of the witness. The competency of an expert is in every case a relative one, i.e. relative to the topic about which the person is asked to make his statement. Wigmore has classified 'occupational experience' as the kind which is obtained casually and incidentally, yet steadily and adequately, in the course of medical practice. The physician must have had basic educational and occupational training as a foundation for his testimony, but it is a practical knowledge that is of controlling importance in determining the competency of a witness to testify as an expert.[5]

In a California case plaintiff sued an orthopedic surgeon to recover damages for injuries alleged to have resulted from malpractice. Plaintiff called an expert in internal medicine who conceded that he had no experience in orthopedics or in any other branch of surgery. Patients coming to him requiring treatment in orthopedic surgery were referred by him to specialists in that branch of medicine. He testified he had some knowledge of what was the proper procedural treatment of such cases from what he had read or from what he had heard from experts in that line. The trial court very properly excluded the witness as incompetent, saying: 'Since the question involved was whether the defendants had used proper skill and care in performing the several operations on plaintiff's foot, the testimony of an expert in internal medicine would be no more persuasive than that of a layman who had read and heard what was the proper professional practice'.[6]

Medical experts should hold in mind that their part in a criminal or civil trial is to aid in the administration of justice. Their opinion is sought and admitted as evidence because it is bottomed on the principle that there are many facts and deductions inferable from facts which are outside the scope of knowledge of men generally. Medical experts need not accept employment as witnesses except upon terms agreeable to them. They need not engage to stultify themselves or to prostitute their profession. They should not engage as an advocate, because no matter how honest the physician, the moment he takes a partisan position, his mode of thinking precludes the presentation of an unbiased opinion.

Satisfactory standards of expertness can be established by trial judges, but the integrity of the medical witness is established by a steadfast course of non-partisanship in medical testimony. Dr. J. W. Courtney, addressing the Harvard class of 1915, said:

'It is the bounden duty of every man in the profession so to shape his conduct toward cases which promise to eventuate into court proceedings, that due respect will be given his opinion, that he will not merit the biting sarcasm, the sneers, the raillery and general brow-beating of opposing counsel. And most of all, that through his efforts the ends of justice will really be accomplished.'[7]

Physicians who embrace Dr. Courtney's counsel will not be subjected to the leers and jeers and sneers of opposing counsel.[8] Biased, partisan medical witnesses may richly deserve it. The partisan witness tends to be argumentative, arrogant, brash and truculent and wholly unwilling to concede the slightest point. 'For him no Minstrel raptures swell.' (Scott, *The Lay of the Last Minstrel.*) I recall a physician who would grudgingly concede that gruel was better for a year-old infant than arsenic.

The expert medical witness can gain much good will by lending a helping hand to the lawyer who may use incorrect terminology. A polite correction should be made in the interest of telling the whole truth. Lawyers frequently confuse the words 'natural' and 'normal'. For example, autopsy findings showed a partial closure of the left coronary artery and an infarction of one-fourth of the left ventricle. There was no history that the deceased had had a prior episode or had consulted a physician. He had worked steadily.

Counsel: Does the autopsy finding show that death resulted from normal causes?
Physician: No.
Counsel: Did Mr. X suffer from heart disease?
Physician: No.

Each answer is accurate but not honest. The entire truth required the physician to say he died from natural causes and he had advanced coronary artery disease.

Lawyers frequently use the words 'symptom', 'finding' or 'diagnosis' whether they are referring to a patient or an autopsied body. Fairness demands that the physician substitute the correct word in his answer and give the entire truth.

In another case the autopsy findings were extensive atherosclerosis of the coronary vessels and aorta. The right coronary artery was almost completely occluded; myocardial infarction.

Counsel: Did the deceased die from an acute coronary occlusion?
Cardiologist: No.

Of course the deceased died from a myocardial infarction and the cardiologist should have said so at once. It was finally dragged out of him, and counsel felt that he was dealing with a sharpy. More important perhaps is the fact that the jury reacts favorably when a physician conducts himself

in a courteous manner. Trial lawyers know that courtesy is essential to success and to-day courtesy toward the medical and lay witness is the rule.

Questions calling for the opinion of a medical witness must be worded so as to require the witness to give his opinion with reasonable certainty. Examples:

Have you an opinion which you can state with reasonable certainty as to the cause of Helen Roe's death?

Have you an opinion which you can state with reasonable certainty as to whether or not injuries sustained by Robert Kenney are permanent and progressive?

The reason why the opinion should be given with reasonable certainty is to preclude speculation, doubt and guesswork. The use of words such as 'possible', 'likely', 'might' or 'could' are quite indefinite and in no sense approach the standard of reasonable certainty. Reasonable certainty is actual certainty which the witness is conscious of after going over in his mind the facts presented and giving consideration to all of the facts.[9] When an opinion is requested to be given with reasonable certainty, an opinion that his condition might be permanent or is very apt to be so is a far cry from certainty. When an opinion is requested to be given with reasonable certainty, the physician should not talk about consequences which are merely possible, or might, or are apt to be. He should avoid loose thinking and weasel words. If he cannot speak with reasonable certainty, he should simply say that he can express no opinion.

In an Oklahoma case the issue was whether Mr. Y died solely from injuries sustained by accidental means or whether disease caused him to fall or caused or contributed to his death.[10] The autopsy findings were severe arteriosclerosis involving the major arteries of the heart; the left coronary artery was completely occluded. There was a superficial cut over the right eye. There was no skull fracture and no hemorrhage into the brain. There was evidence that Mr. Y had fallen forward to the sidewalk. He was pronounced dead on arrival of an ambulance twenty minutes after the fall. From this evidence and the autopsy findings a physician gave his opinion that Mr. Y died from natural causes and that it was probable that his heart condition caused him to fall. On cross-examination he was asked this question and gave this answer: 'Q. Now, if this man had this condition that you've found there, and he did have a fall or there was some shock to his entire system, wouldn't that possibly accelerate that condition and bring it to a focus? A. It is certainly a possibility. In other words, I couldn't say that such a thing is absolutely impossible. I consider it very unlikely.' The answer is a cogent example of slipshod thinking. Merely because counsel inquired about the possibilities of the case did not require the physician to abandon thinking and talking with reasonable certainty about the facts of the case. The question called for a 'yes' or 'no' answer. The answer should have been, 'I cannot answer that question without an explanation.' If counsel was unwilling to accept an answer with an

explanation, the physician should have let his answer stand. An answer that a question cannot be answered 'yes' or 'no' without an explanation is responsive and is often the only one that can honestly be given. If counsel indicated willingness to accept an answer with an explanation, the physician could have said, 'In my opinion he died solely from natural causes. I would only be guessing if I talked about possibilities and my guess would be no better than that of any layman.' When a physician is called upon to express an opinion he should think and talk with reasonable certainty about the facts. He should eschew possibilities and speculations.

In some states[11] the question must be phrased in this way:

'Have you an opinion which you can state with reasonable certainty as to whether there *might* or *could* be a causal relation between injuries sustained and the death of John Winter?' The reason given by some courts why the physician is restricted to a 'might' or 'could' answer is that, if he were to testify that there *is* a causal relation, he would invade the province of the jury. This rule has not the slightest basis in reason. The opinions of medical experts about the cause of disease or death or the causal relationship between accident and injury or injury and death do not invade the province of the jury. Such opinions are given to the jury to be considered and weighed with all the other evidence on the issue of causation. When medical knowledge is necessary to enable a jury to determine the cause of death or disease, an opinion that a certain cause did or did not produce a certain result in no sense impinges upon the jury's right to determine the issue of causation.

Courts that require a medical opinion to be given with reasonable certainty and at the same time insist that the opinion be given on a 'might' or 'could' basis invite the witness to be cocksure about a mere possibility. In so far as an opinion is based on reasonable certainty it cannot also be based on a possibility, certainty and possibility being by definition contradictory terms. As Professor McCormick pointed out:

> 'This restriction is unfair to the expert. If he answers *yes*, the jury may believe that his opinion is no stronger than a belief in the possibility of causal relation, whereas in fact the doctor may be convinced that the accident *was* the cause, but is not allowed to say so. Moreover, if he should answer *no*, it could not have been the cause, the answer would offend the court's canon, for it would clearly be an opinion on the issue.'[12]

When a physician is unable to give an opinion about which of several causes was the reasonably certain cause of the condition, he should promptly say so. His candor will favorably impress the court and jury, and an adept lawyer will soon expose him if he does otherwise.

The purpose of expert testimony is to aid the jury; hence, the medical expert must make himself understood. Big words and technical terms unexplained merely bamboozle a jury. In *Prudential Ins. Co. of America* v. *Lowe*[13] a physician was asked to describe the condition which he found in a layman's language. He did it in this way:

We found a schirrous adenocarcinoma of the small intestine with metastasis to parietal peritoneum and serosa of the greater part of the intestinal tract. Adenocarcinoma is a type of cancer. We also found multiple obstructions of the small intestine due to tumor infiltrations and adhesions. There was a double enterostomy opening near the midline with a healed rectus incision on either side. The court remarked, 'This simple language was for the benefit of laymen.'

It is time to bring these hurried and crowded remarks to a close. The field of expert medical testimony offers a challenge to the greater efforts of the members of two great professions. Gentlemen, I say to you: 'Be noble yourselves and the nobility that exists in other men will rise in majesty to meet you'.

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2. Masergian *vs.* Candell, 214 App. Div. (N.Y.) 730.
3. Wigmore (1923): Volume **1**, Section 563.
4. *Medical Expert Testimony*, Handy, L. S. American Life Convention, 1940; *Medical Testimony in Personal Injury Cases*, Elliott & Spillman, Law and Contemporary Problems, October, 1935.
5. 2 Wigmore Evidence, 3 Ed., Sec. 556; Sinz *v.* Owens, 33 Cal. (2d) **749**, 205 P. (2d) 3.
6. Pearce *v.* Linde, 113 Cal. App. (2d) **627**, 248 P. (2d) 506.
7. Boston Medical and Surgical Journal, January, 1916.
8. *Suggestions for Improving Medico-legal Court Procedure*, **24** Amer. J. Surg. (N.S. 199).
9. Walker's Case, 243 Mass. 224, 137 N.E. 171; Wallace *v.* Ludwig, **292** Mass. 251, 198 N.E. 159; Rayner *v.* Metropolitan St. Ry. Co., **106** App. Div. 449, 94 N.Y.S. 632; Gleason *v.* Hudson Valley Ry. Co., **143** App. Div. 884, 128 N.Y.S. 758.
10. Equitable Life Assur. Soc. *v.* Neale, (Okla.) **258** P. (2d) 654.
11. Fellows-Kimbrough *v.* Chicago City R.R. Co., **272** Ill. 71, 111 N.E. 499; DeHaan *v.* Winter, **258** Mich. 293, 241 N.W. 923; Patrick *v.* Treadwell, **222** N.C. 1, 21 S.E. (2d) 818.
12. Louisiana Law Review, March 1952, Page 265.
13. **113** Ky. App. 126, 230 S.W. (2d) 466.

Evidence of Blood Test Results

Sidney B. Schatkin *
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The New York blood test statute (New York City Criminal Courts Act, Section 67, subd. 1-a) provides as follows:

> The Court, on motion of the defendant, shall order the mother, her child and the defendant, to submit to one or more blood-grouping tests by a duly qualified physician to determine whether or not the defendant can be excluded as being the father of the child, and the results of such test may be received in evidence but only in cases where definite exclusion is established.

Only the defendant may demand the test, the result of which is admissible in evidence only when there is an exclusion. The test can aid the defendant exclusively and, obviously, the defendant will be the party offering in evidence the 'definite exclusion'.

There are certain unusual blood test results that the defendant will be tempted to offer in evidence. For example, to suppress a blood test result which indicates a high degree of probability of non-paternity, bordering on certainty, seems a miscarriage of justice. Conversely, the mother's inability to offer in evidence a blood test result which constitutes cogent circumstantial evidence of actual paternity, seems equally harsh. For example, in one otherwise inconclusive result, both the accused man and the baby were Kell positive and Dr. Wiener noted that 'The Kell tests indicate the probability of paternity, since Kell positives are only 8 to 10 per cent.' Dr. Wiener's observation was, of course, inadmissible in evidence, but the Court was deprived of knowledge of that item of strong circumstantial evidence. It seems that the Courts are not yet ready for circumstantial evidence in connexion with blood tests.

In *Miller* v. *Domanski*, 97 A. 2d, 641, the defendant's appeal from a judgment of paternity was predicated chiefly on the Trial Court's refusal to permit Dr. Levine to testify for the purpose of qualifying an otherwise inconclusive blood test result. Dr. Levine conceded that the test did not exclude paternity, but he wanted to testify concerning 'an assumption that the mother's blood has a very rare and unusual serologic makeup'. This the Court did not permit, and allowed the inconclusive result to stand, without qualification. Dr. Levine stated:

> 'Because of a very unusual serologic finding, I could not use my usual report. Where there is no exclusion of paternity, there is a qualified serologic detail, which I will disclose if I have the opportunity. . . . *The tests in this case would show no exclusion, but we have to make the assumption that the mother's blood has a very rare and unusual makeup.* If she has a usual serologic makeup, why then, it would have to be considered an exclusion.'

Dr. Levine clearly was attempting to explain to the court that the blood

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test result constituted strong *circumstantial* evidence of non-paternity. This he was not permitted to do.

To justify its rejection of this evidence, the Court noted that admissibility in evidence is confined to exclusions only—solely as evidence of actual non-paternity. The Court went on to state:

'To permit Dr. Levine to testify that an "assumption that the mother's blood has a very rare and unusual serologic makeup", attempting thereby to explain away the failure to attain a negative result in the tests, would be to admit the testimony of additional scientific facts not available in that field in 1939, when the statute in question was adopted, and thereby impel judicial recognition thereof contrary to the legislative expression of public policy, or would constitute a mere attack upon the reliability of exclusionary findings in the tests . . . blood grouping tests are admissible as an evidential aid only for the limited exclusionary field referred to. . . . In view of the fact that Dr. Levine testified that "the tests in this case show no exclusion", we conclude that there was no error in the Court's ruling.'

The affirmance was by an Appellate Court divided 2 to 1. The dissenting justice grasped what was agitating Dr. Levine, for he observed in his dissenting opinion, 'that in some instances at the present time, the conscientious expert can speak only of probabilities and not of absolutes; he can give his opinion but not a categorical answer. . . . The present case seems to have been one of those instances. The Court should have questioned Dr. Levine further in order to bring out clearly what he meant—even though thereby the doctor's opinion on the question of paternity might have been disclosed.' The dissenting justice further significantly observed:

'Does the statute, L. 1939, c. 221, prevent the reception of the evidence? When enacting it, the Legislature understood correctly that blood grouping tests could not prove that the defendant was actually the father of the child. And undoubtedly the Legislature feared that if the expert were allowed to testify that the tests did not exclude the defendant, the negative might be accepted by the jury as a circumstance corroborating the charge that he was the father; so they enacted that the expert's testimony should be received only "in cases where definite exclusion of parentage of the defendant is indicated."'

That observation by the dissenting justice touches the heart of the matter. The real and only reason why the legislature provided for admissibility in evidence only where the test excludes, was to *prevent a complainant from using an inconclusive result against the defendant.* The legislature did not intend to bar a defendant from using a test result which *tends* to exclude, although not an absolute exclusion.

However, the Courts are not yet ready for this innovation; and it is perhaps preferable at this time to avoid controversy where blood tests are concerned. In the course of time, when the courts are ready, the law will undoubtedly realize the great value of certain blood test results as circumstantial evidence of non-paternity, and even actual paternity. Until that time is reached, however, the serologist must confine himself to a report indicating possibility or absolute impossibility of paternity, without further qualification.

Where the accused man and the twin children possessed the rare type rh″, Dr. Wiener noted, 'the accused belongs to a very rare blood type rh″ which occurs in only 1 out of 200 individuals, and since both twins also belong to type rh″, this is rather strong circumstantial evidence that he actually is the father of the twins'. Such evidence is inadmissible.

Similarly inadmissible was the strong circumstantial evidence of paternity where defendant and the baby both possessed the rare rh^w factor.

And where the accused man was Rh_1Rh_2 and the baby rhrh, Dr. Wiener observed:

> 'Although the tests do not exclude the defendant, the combination of Rh types is very unusual and could only be explained by assuming that defendant possesses the rare gene R^z. The rarity of this gene may become clearer if I explain this as follows: In 25 cases like this one, only one of the men would prove to be a carrier of the R^z gene, while the other 24 men would turn out actually not to be the fathers.'

Though Dr. Wiener may have been reasonably confident, as a practical proposition, that paternity was excluded, the legal certainty of definite exclusion is lacking, and hence Dr. Wiener's views are inadmissible in evidence.

As already noted, Mr. Justice Bigelow, dissenting in *Miller* v. *Domanski*. 97 A. 2d, 641, touched the heart of the matter when he observed that the legislative intention in making admissible only a definite exclusion of paternity, was to prevent the mother from using an inconclusive result against the alleged father. The legislature did not seek to prevent the defendant from offering in evidence a test result which constitutes circumstantial evidence of his non-paternity.

There is reason to believe that in time Justice Bigelow's view of the matter will become the prevailing opinion.

Vast scientific strides in the field of serology have been made since the enactment of the blood test law in New York in 1935. That scientific progress will ultimately be reflected in adequate legislation.

In the meantime, we know that judicial acceptance of blood tests is largely due to the fact that the law considers blood tests non-controversial. The law accepts the simple answer of Science: 'Yes, he may be the father' or 'No, he cannot be the father'.

At this time, to inject elements of controversy, or 'opinion' or circumstantial evidence in medicolegal blood tests, will create a doubt, in the court's mind, of blood tests, the complete reliability of which is now fully accepted by an informed court.

In time, however, blood tests, with all their potentialities and possibilities, will become known to, and accepted by the Courts.

For the present, therefore, let the serologist confine himself to a report which either definitely excludes paternity, or is inconclusive on that point.

Hemolytic Transfusion Reactions

Medico-Legal Aspects‡

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Blood is a biologically active substance of therapeutic value. It is a perishable product which must be derived from human beings in thousands of laboratories widely scattered throughout the country. Nothing administered to patients is potentially so dangerous as mishandled blood. For this this reason all possible precautions must be taken to insure safety.
Hemolytic transfusion reactions may result from one of several causes:

1. Contaminated blood.
2. Overaged blood.
3. Mechanical defects in the transfusion procedure.
4. Antigen-Antibody reactions in vivo.

1. Contaminated Blood

Human blood collected in anti-coagulant solution offers an exceptionally good medium for the support of many bacteria. Emphasis must be placed on the collection of blood from healthy donors only, in order to avoid the transmission of disease from one person to another. In addition, it is imperative that all precautions be taken to eliminate the risk of contamination of the blood collected during the bleeding process. It should be unnecessary to emphasize the need for care in the preparation of the equipment used in collecting the blood. Blood after collection must be handled in such a way as to discourage the growth of any chance contamination which may have avoided the barrier set up against contamination of blood. The operation of refrigerators should be closely watched to ensure the proper storage temperature. Refrigerators, however, cannot be relied upon to prevent the growth of certain organisms which may be capable of producing jaundice or hemolysins which may be extremely dangerous to

‡ A paper read at the Annual Meeting of the American Academy of Forensic Sciences in February, 1954.
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patients. The practice of culturing all filled blood bottles might result in a greater danger of contamination during the culturing process than exists in the collection of the blood. It therefore becomes imperative that technics be faultless in regard to the selection of the donor, the cleansing of the bleeding site, the use of sterile equipment and the proper storage of blood.

Reports of recipient reactions due to bacterial contamination are not as frequent as the event itself. Reactions due to bacterial contamination are usually characterized by chills and fever and the laboratory findings associated with the rapid breakdown of red cells, i.e., hemoglobinemia and hemoglobinuria associated with a marked increase in bilirubin I. The reactions may be followed by shock, oliguria or anuria, and in some instances by death of the patient.

Technics for the collection of blood are a responsibility of the laboratory and any negligence in regard to the proper handling of blood would be extremely damaging to the laboratory.

2. Overaged Blood

Increased demands for blood during World War II resulted in the development of solutions designed to afford maximum preservation of human red cells. The red cells of human blood collected are of many ages and it would seem reasonable to assume that aging processes of cells in preserving media continue after the blood is collected. It has been established that whole blood collected in ACD mixture will have therapeutic usefulness for a period of 21 days. Beyond that time the aged red cells will be increasingly less stable. Occasionally stored blood exhibits hemolysis and such cells are more readily broken down following transfusion. Jaundice resulting from such a transfusion must be distinguished from that of the more serious antigen-antibody reaction. If the blood transfused is also pyrogenic, it is absolutely necessary to assume that the fever and jaundice is due to an antigen-antibody reaction and studies initiated immediately to rule out the latter possibility. The consequences of transfusion of overaged blood fortunately are not severe but the patient does not derive the expected benefit from the transfusion; and, from a medical-legal standpoint, the recommended limits on storage of blood must not be exceeded. Likewise blood should be inspected for evidence of hemolysis in the supernatant plasma. Where this phenomenon is evident, the blood should not be issued.

The maintenance of proper storage conditions for blood are often thought to pertain only to the maximum allowable temperature. However, not infrequently mechanical refrigerators may become too cold for the proper storage of blood. Whole blood which reaches a temperature permitting the formation of ice in the bottle will usually result in hemolysed blood, which should not be issued to patients.

3. Mechanical Difficulty in Transfusion

With the increased use of blood and the relative ease with which the staffs of hospitals can obtain and administer blood, there is an increasing tendency to regard blood transfusion as a minor medical procedure. For example, a whole blood transfusion was started by a nurse trained in intravenous work and the blood was allowed to run into the patient without any observation. A considerable portion of the contents of the bottle was deposited into the soft tissues of the patient's arm since the needle did not stay in the vein. This extravascular blood was rapidly broken down and the following morning the patient was jaundiced. The laboratory received a report of jaundice and exhaustive studies of the patient's blood with that of the donor revealed no incompatibility. The nature of the untoward reaction was found on a visit to the patient. The badly discolored arm and the temporary increase in bilirubin I, made it apparent that the source of jaundice was the extravasated red cells. Serious complications did not result since the patient promptly cleared the excess amounts of pigment. In this instance the fault and responsibility lay with the individual who failed to supervise the transfusion procedure properly. Blood transfusion is an important therapeutic procedure which, because of its inherent danger, must be properly regarded by those who administer blood.

4. Antigen-Antibody Reactions

The increased use of blood to correct human physical disorders is associated with an increasing risk because of the individuality of human red cells. The increasing frequency with which individuals receive multiple transfusions and the growing knowledge of the antigenic character of human red cells calls for more intelligent and careful measures in laboratories to prevent serious damage to patients and preserve the life-saving qualities of human blood.

As in all medical procedures, there is an obligation to conform to local standards of practice and in view of our present and changing knowledge concerning blood antigens and antibodies, the standards cannot be static. They must be raised in order to afford maximum protection to the patient. The concept of standard practice in blood transfusion has undergone great and rapid changes since the description by Wiener and Peters of intragroup transfusion reactions in 1940. A great avalanche of data has accumulated concerning the immunization of man by blood antigens caused by blood transfusion or by pregnancy. We have been privileged to hear of some of the newer clinically important blood factors to-day and no doubt at the next general session of this Academy there will be some factor unknown to-day, which will be described as a cause of immunization to man.

Some laboratories appear to be unable to keep abreast of knowledge

as it accumulates. Eight years after the original report that Rh antigen and its antibodies were capable of producing hemolytic transfusion reaction, in a large city in this country, an attempt was made to establish that determination of the Rh type of the donor and recipient was not required to conform to local standards of practice. The case was settled out of court after it was pointed out for how long a period of time Rh-typing reagents had been commonly available and that knowledge was sufficient to make it a requirement for blood transfusion.

The human red cell is capable of carrying numerous distinct and specific antigens and many have thus far been described and proven to cause immunization in man. Nevertheless it is probable that many more antigens will be found although the frequency with which they immunize man is not expected to approach that caused by the Rh_0 (D) factor.

The discovery that Rh antibodies do not necessarily agglutinate red cells suspended in physiological saline affects the development of more inclusive technics for demonstrating *in vitro* reactions of blood antigens and their antibodies. It is reasonably well recognized that a crossmatch containing the serum of the recipient and saline suspensions of the donor does not afford maximum protection to the patient. The development of the high protein crossmatch served a great usefulness in our laboratories. Recently, because of a failure of the high protein crossmatch to disclose an incompatibility due to the Kell factor, which resulted in a hemolytic transfusion reaction, our laboratory changed the practice of indirect Coombs test crossmatching on selected recipients only, and made it a routine procedure for the major side of all crossmatching.

This incident of Kell immunization was helpful in that it was found that a simple slide test procedure in which the serum of the immunized recipient plus whole blood from the donor mixed and placed on a warm illuminated box, gave rapid and massive clumping. Agglutination did not take place in test tubes in which the same serum was mixed with cells suspended in saline and 30% albumin added. The simplicity of the slide test procedure together with the demonstrated ability of the high protein crossmatch to detect Rh-Hr and blood group incompatibilities, gave an additional and feasible safeguard to the recipient. The indirect Coombs test procedure before this incident was performed selectively on (1) individuals who had received multiple transfusions in the past; (2) those who had reports of pyrogenic reactions during prior transfusions; and (3) on women who were pregnant and who had borne children. The conditions for preventing the hemolytic transfusion reaction due to the Kell immunization cited here were in operation, but unfortunately the blood bank was not told that the woman was pregnant or that she had had previous children. Either the slide test or the Coombs test would have been capable of demonstrating the incompatibility had it been done at the proper time. The woman suffered a severe hemolytic reaction.

The indirect Coombs test requires a period of incubation of not less than 30 minutes for sensitization of the red cells and about 10-15 minutes for careful washing of the red cells before the test is performed. In the emergency, such delay may be impossible. We have adopted the procedures of the *slide test,* the *high protein tube* crossmatch and the *indirect Coombs* test crossmatch. If the situation warrants the issue of blood before the Coombs test is completed, the blood is issued from the blood bank and the procedure continued. If incompatibility by the indirect Coombs test is found, blood may be intercepted before it is given. It is not expected that this will be a frequent happening since more than 7,500 crossmatches thus far have been performed with the indirect Coombs test method and no incompatibility has been detected which was not apparent by the other two tests.

In our laboratories we have adopted a ritualistic procedure in performing the crossmatch. We have designed a small crossmatching block which will contain specimens from one recipient and one donor only. The procedure must be completed with each bottle before a second bottle is begun. This is intended to prevent serious errors in crossmatching due to confusion of specimens.

Great emphasis is placed on the proper demonstration of the blood group of the donor and recipient since the major cause of incompatible transfusion reaction remains, as would be expected, in blood grouping where antibody is almost universally present. In addition, a high protein crossmatch is performed on the major and minor side. A simple slide test procedure using recipient serum and donor whole blood is performed on a viewing box and the indirect Coombs test completes the crossmatch.

Thus, improvement in technic, and in particular in the broadening of the range of safety of the crossmatch, will give the laboratory means better to serve the patient and less cause for the concern of charges of negligence or incompetence.

In fairness to the laboratory, we must cite our own experience of approximately 125,000 crossmatches during the period 1949-1954. There have been 5 instances of hemolytic transfusion reaction; 3 were due to ABO incompatibility—all resulting from failure to read the label on the blood bottle by those who administer blood; two were errors of the laboratory—one due to Rh_0 blocking antibody following which high protein crossmatch in tubes was devised (1947). The second laboratory shortcoming was due to Kell immunization (1953) leading to *routine Coombs* test crossmatching. Many dangerous instances have been averted by our alert staff.

Blood has no margin of safety if mishandled—in the donor room, laboratory or in surgery—or at the bedside of the patient. Knowledge must be assimilated and used—negligence will not be tolerated by patients and their families.

Some Medico-Legal Aspects of Aggression†

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Aggression is an expansive term. From a broad viewpoint, the aspects of aggressive or self-assertive behavior range all the way from the cries of a new-born baby to the attempts of would-be conquerors to rule the world. Elementally, it is universal and ever-present in life and nature. Aggressiveness in its benign form is essential to self-preservation and social adaptation. It becomes dangerous and destructive only when it swells into an abnormal flood tide and overflows the channels that control its usefulness. Like the atom, the modern symbol of protean force, it can be harnessed for benefit or unleashed for ruin.

Consider the multitude of guises in which some trace of aggression may be recognized. These may be said to include children's games, sibling jealousy, youthful unruliness or delinquency, lovers' quarrels, domestic discord, spectator and participating sports, card-playing and gambling, the driving of automobiles, satirical humor, the reading of detective stories, reformism, street fights, strikes, race conflicts, lynching, suicide, crime and war.

As Sir Thomas Browne observed long ago, 'man parallels nature in the cosmography of himself'. There is hardly a phase or mood of nature that does not have a corresponding attribute in human existence. Birth, growth, integration, accommodation, progeneration and decay—these we have in common with our milieu. And just as the face of nature varies from the gentleness of springtime to the angry storms of winter, in keeping with its processes of maintenance and renewal, so the behavior of man fluctuates from congenial cooperation to stern competition and struggle for life's prizes.

The label of aggression is not always reprehensible. Only when it goes to extremes and escapes the bounds of socially accepted standards does aggressiveness become troublesome.

Proverbially, human nature has a dichotomy or ambivalence that presents

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two faces, or what were once regarded as two souls: one of sociability and tolerance, the other of jealousy and antagonism. When a strongly desired goal is visualized, man strives to attain it by any means available to him. The interaction of the contending forces of the personality is such that if the means of attainment are inhibited by reason of law or custom or conscience or a superior counter-force, then the defeat or deprival amounts to a frustration. Some individuals resolve their frustrations by philosophical means, divert them to other channels, or sublimate them; but not everyone is able to do so.

Aggression of the destructive kind usually connotes a low threshold of frustration tolerance. Deprived of satisfaction for a strong impulse, persons deficient in this tolerance may react in a manner that leads to so-called criminal conduct. The potentialities for archaic or primitive outbursts lie more or less deeply buried in every personality. These explosive kernels of atavism can be evoked and implemented by the aggravations of frustration, more easily so in some persons than in others.

From this viewpoint, violent or unsocial conduct cannot be attributed to tendencies that the law regards as vicious. It is not uncommon for individuals suffering from some forms of epilepsy, for example, to engage in violent episodes without any conscious knowledge of their actions. There are persons, too, whose perceptions are so dim and their self-direction so inadequate that they may succumb to aggressive impulses without being overtly criminal or demonstrably irresponsible.

One may cite the case of a man who is now under hospital care. He came back from war service out of tune with his environment. His inability to control his aggressive tendencies led him into one scuffle after another. He picked fights in the street and got into bar-room brawls. He broke windows and damaged other property during outbursts of temper. He was continually at loggerheads with someone or something. Eventually, to escape the legal consequences of his tantrums, he voluntarily entered a mental hospital. Even there his aggressive bent continued. He would not undergo any recommended systematic treatment. Embittered by his grudge against the world, he repeatedly gave rein to jealous resentment toward anyone around him who appeared to have even the smallest kind of ability or success. Once he engaged in so violent an altercation with a hospital employee that he tried to gouge the man's eyes out in the commando manner, hit him with full force in the stomach and would have beaten him with a fireplace tongs if he had not been fortunately restrained by the alertness of other personnel. Yet this man is not commitable. If he were outside an institution he would have been arrested and penalized for assault. Even if serious consequences had occurred in the institution, he would not have been saved from the severity of legal retribution.

What are we to do with such persons? From the medicolegal viewpoint, the resolution of this dilemma is a timely problem for forensic medicine.

These are not even borderline cases. They constitute an intractible no-man's-land between the codified formulas of jurisprudence, one one hand, and the terminology of recognized psychiatry, on the other. These people have an ability to converse and behave in a manner that gives a favorable impression to the superficial lay observer. To the professional observer they might appear to belong to the outmoded psychopathic classification, to which, in reality, they show no other true resemblance than clothing aggression. Their intellect is unimpaired; at times it is rather high. Their hostility is so deeply buried that it requires a forceful emotional impact to bring it to the surface, but then a cyclonic force carries them to great extremes, without ability to stop or moderate their fury. If left to their own devices, they will inevitably be swept into the maelstrom of violence that will carry them into penal institutions.

There is the dilemma: people in this category cannot be held fully responsible from the medical viewpoint, and yet they cannot be exempted from the legal viewpoint.

In legal parlance, crimes committed in hot blood are vested with some of the ameliorating circumstances that are commonly weighed in the scientific disposal of man and his problems. In the cases under discussion here, we are unprepared and not equipped to deal with them from a detached scientific viewpoint. So we must deal with them from the emotional viewpoint by allowing the ameliorating circumstances to be considered.

I should like to see a judicious arrangement under which each person brought up as a consequence of aggression would be legally compelled to undertake psychiatric hospitalization. The old-fashioned device of immuring such offenders in correctional institutions not only makes their effective adjustment impossible, but impairs their capacity for adjustment in proportion with the length of time they are compelled to serve.

Legislation is needed for the proper disposal of this category of cases. This would not necessitate the usual legal commitment for mental incapacity, but would empower the judge, with the assistance of his psychiatric experts, to exempt the hospital authorities from obtaining the usual permission required from the patient, for pharmacodynamic or physiological treatment of the offender, if he is unsuited or unwilling for psychotherapeutic procedures. These, of course, have to be conducted on a voluntary basis.

A procedure of this kind would result in a wide range of benefits. It would modernize court methods with respect to this field, with gains in economy and effectiveness. It would help this category of patients to combat their difficulty, instead of only deepening it through imprisonment. And it would afford a valuable opportunity for extensive observation and research on the genesis and control of the many varieties of aggressive behavior.

Morphological Findings in Some Endogenous Mental Disorders

Their Value in Forensic Pathology

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In a recent lecture on *Suicide and Insanity, as Seen by the Pathologist,* we pointed out how limited is the value of our 'potential' micro- and macroscopical findings. While our discussion was aimed mainly at medical laymen (judges, criminologists) with the goal of convincing them of our inefficiency in this respect, it is the purpose of this paper to review the subject for the pathologist. We are not in a position to bring new evidence but intend only to refresh the reader's memory with the aid of a basic compilation. That our subject is of importance can be borne out by the fact that the question often arises whether a will should be contended, if followed shortly by unexpected death or whether an alleged crime was committed by an insane person who succumbed afterwards. In other words, the pathologist is sometimes asked whether or not the deceased was sane before his death.

In older textbooks on pathology and forensic medicine one may read the statement that opacity or thickening of the dura matter is a manifestation of premortal insanity. These findings are of no importance in this respect, as they may also be met with in deaths from syphilis and acute infectious diseases. Retrospective investigations in a large number of these cases did not reveal permanent or transitory insanity during life.

It would go far beyond the space allotted to discuss all mental disorders in which gross and microscopic changes may be seen. Those which may offer somewhat typical histological findings are numerous and their pictures are well known (general paresis, vascular diseases, certain infectious diseases, primary and secondary tumors). Here, we are only interested in discussing a certain group of endogenous mental disorders which *a priori* lack characteristic findings, but which may present potential findings. We are referring to certain types of symptomatic epilepsy, different types of alcoholic psychosis like delirium tremens and chronic alcoholism, endogenous psychoses like idiopathic (true) epilepsy, schizophrenias of different types, manic-depressive psychosis and involution psychosis. Do all these psychoses have certain findings in common, apart from occasional petechiae, and, if there are special findings how pathognomonic are they for a certain psychosis? There are some findings common to all these psychoses, but they are not very numerous and are not characteristic of any single disease. In this respect we refer to cerebral edema and cerebral (dry) swelling, changes which may be found sometimes as the only manifestations in cases of cerebral death (central death).

In cases of *cerebral edema* the brain is enlarged, its weight is increased and the tissue is permeated by fluid. Such a brain occupies the cranial cavity completely, the gyri are flattened, the sulci are indistinct, the olives and the tonsils of the cerebellum are more distinct than usual and, due to their increased volume, they are forced into the foramen magnum (pressure cone). The ventricles are slightly dilated and contain a moderately increased amount of cerebro-spinal fluid. The brain's cut surface is bulging, wet and glistening. The consistency of the organ is decreased and cross sections of the small blood vessels offer rather characteristic findings. These vascular cross sections, referred to as 'blood dots', can be wiped off easily, contrary to small true hemorrhages originating from small blood vessels, which cannot be removed. In cerebral edema these 'blood dots' dissolve rather quickly, because the escaping blood and the edematous fluid mix readily. In later phases of edema (as mentioned by B. Mueller whose survey on the subject we are following) single areas of the brain become very anemic and exhibit patches, known as *Kolisko's focal pallor.* In cerebral edema we are dealing with an increase of fluid which accumulates between the cells, especially in the white matter. Under the microscope the tissue shows a loose texture due to the large amount of fluid. For the same reason, the perivascular spaces (Virchow-Robin spaces) are distended and the limiting glial membrane is retracted. In the neighbourhood of the vessels, the glial cells are reduced in number.

In cases of *cerebral swelling* the brain is also enlarged and heavier than normal but, contrary to the case in cerebral edema, its consistency is increased, its cut surface dry, no fluid escapes and the tissue is sticky. Sections have the tendency to adhere to the cutting knife and the blood dots do not dissolve as in cerebral edema. The ventricles are empty and narrow but gyri and cerebellum offer similar signs of increased pressure as described in cerebral edema. The basic difference between these two conditions lies in the fact that in cerebral edema the fluid is dispersed between the tissue elements, while in cerebral swelling it is located within the cells and their processes. In cerebral swelling the tissue adherent to the medullary sheath is swollen while the rest of the brain elements are separated. The protoplasmic glia disappears and its processes are broken up. The perivascular spaces are loosened up, especially in advanced cases, as pointed out by Ostertag. Whether this phenomenon is caused by adhesions of edematous fluid, delivered by the blood vessels, not set free, but combining with the brain substance to be changed into a firmer colloidal condition, or whether it is linked with retention of urea (De Crinis), is still under discussion.

Reichardt was the first to describe the picture of dry swelling of the brain and he also investigated, based on his own methods, the relation and differences between the capacity of the skull and the volume of the brain in these cases .He found that the latter is surpassed by the former by about 10% in adults, 5% in small children and about 2.5% in the new

born. As death in dry swelling of the brain is caused by the simple fact that there is not sufficient space for the enlarged brain within the skull, it is evident that the smaller the difference in the single case, the greater the danger of a fatal outcome. In other words, in spite of potentially open sutures in newborns and small children, they are more endangered than others. That pre-existing synostosis of sutures or rickets complicating such swelling, will favor sudden death, is therefore well explained. We know of cases which offer 'a constitutionally low Reichardt index' and if they come to autopsy they present signs of chronically increased cerebral pressure. If the latter is manifest during life, then a slight change or disturbance brought on by minor external events, e.g., fright or psychogenic trauma, may increase the cerebral volume and may cause sudden death. But such a possibility does not entitle us to establish a chain of causation between psychic disturbances and sudden death, as a routine conclusion. Assumption for such a connexion would be justified only by the demonstration of an anatomical basis for chronic increase of cerebral pressure plus an intravital event, in the absence of other findings that might explain sudden death.

According to Peters, B. Mueller and others, cerebral edema may change into cerebral swelling, a possibility sometimes neglected. Furthermore, it cannot be predicted under what conditions we may expect to find edema and when we may expect cerebral swelling. It seems that younger people incline more to dry swelling than older. Neither of the two conditions under discussion is specific (pathognomonic) for one basic pathology.

Cerebral edema is very often associated with different pathological conditions. Cerebral swelling is met with in sudden death in which other autopsy findings are poor or absent, in diphtheria toxemia, sometimes in septicemia, in peritonitis, eclampsia, certain poisonings (barbiturates, lysol), syphilitic vascular disease of the brain, uremia, kidney disease, fatal hemorrhage, strangulation (but not in drowning), electrocution, etc. This enumeration illustrates the limitations of its diagnostic value in forensic pathology, not to mention that of brain and mental disorders. Nevertheless, it must be emphasized that dry swelling is often found in the neighbourhood of brain tumors (Hamperl), after brain surgery, brain concussion, brain abscess, in deaths taking place in status epilepticus and paralyticus, in deaths preceded by catatonic stupor and in the course of schizophrenia. As a rule, one has to agree with Toennis who states that dry swelling belongs more to the realm of the internist and psychiatrist, while cerebral edema is more often seen by the surgeon. The important role of dry swelling of the brain in the course of acute catatonic stupors is discussed by Stoessel.

Summarizing, the anatomical presence of dry swelling of the brain, preceded by psychoses and mental disorders, should be taken at its face value. Only if other findings are absent and a careful clinical history is available has it some limited forensic importance.

Discussing facultative morphological changes in special psychoses and mental disorders, as outlined previously, we shall now start with different types of *alcoholic psychoses,* but shall confine our survey to morphological findings, omitting toxicology. Although acute ethyl alcohol intoxication causes definite injury to the central nervous system, only non-characteristic changes, like hyperemia of meninges and brain together with cerebral edema may be found. If a large amount of alcohol was absorbed by the tissue, the characteristic smell may be noticed at autopsy. Smaller hemorrhages in brain and ventricles may be present, but extensive or even moderately large intracerebral hemorrhage should be attributed to alcohol only if pre-existing vascular lesions can be excluded. This is of forensic importance, because alcohol raises the blood pressure which may, in turn, lead to rupture of a sclerotic vessel. Therefore a pre-existing lesion as a potential extenuating circumstance is a possibility to be explored in each case. The findings under the microscope in acute alcoholic intoxication may be only non-specific changes in nerve cells, like destruction of Purkinje cells. The latter was also found in experimental alcoholic intoxication. Sometimes alcohol may be found in the spinal fluid (Schottmueller). Acute alcoholic intoxication *per se* is rarely fatal. If so, it is mainly due to superimposed pathologic changes, like injuries, aspiration pneumonia, rupture of esophagus (Flipse), etc.

Chronic alcoholism may become manifest anatomically in different ways. Lesions may be found in the brain, spinal cord and peripheral nerves, but again we have to emphasize that they *may* be found and that they could be noticed in grey as well as white matter. Thickening and opacity of meninges, cerebral atrophy plus internal hydrocephalus may be found, but concommitant senile and vascular processes have to be excluded before the changes are attributed to alcoholism alone. A few petechiae are sometimes found, usually in the region of nuclear masses, adjacent to the ventricles or around the oculo-motor nuclei (A. B. Baker). More about this condition, also known as *Wernicke's polioencephalitis hemorrhagica superior,* (*Wernicke's encephalopathy*) later on. But there are many cases in which such gross findings are completely absent. Following the impressive outline of the subject by Peters we should like to mention that numerous histological changes are known to occur in the ganglion cells, such as fatty change, vacuolization, shrinkage and demyelinization of neurites, but none is pathognomonic for chronic alcoholism *per se.* Changes in the corpus callosum claimed by Marchiava, Rossi and others as characteristic of alcoholism consisting of demyelinization, accumulation of fatty granules and glial proliferation with preservation of axis cylinders are of major importance only in the presence of a clinical history and the absence of other lesions. The presence of cirrhosis of the liver makes a decision easier, but even hepatic cirrhosis is by no means always of alcoholic origin (e.g., cirrhosis occurs in the Bantu due to abnormal food and vitamin deficiencies).

Another potential manifestation of chronic alcoholism is the already mentioned *acute superior hemorrhagic polioencephalitis of Wernicke.* These changes also occur in other conditions, like Pb, Mn, CO and H_2S poisoning and in Wilson's disease. The areas of predilection are, according to Peters, the vegetative centers of regulation around the third and fourth ventricle and the aqueduct, corpora mamillaria, the oculo-motor centers and the dorsal part of the vagus nucleus, the nerves supraopticus and paraventricularis. This topographical distribution is in accordance with clinical symptoms collected under the term Korsakow's psychosis. Though sometimes absent, the corresponding gross changes may be recognized and may consist of sclerosis of the mamillary bodies which may exhibit a yellowish-grey color associated with hemorrhagic dots. Histologically, we may find circumscribed proliferation of connective tissue, new formation of blood vessels, glial proliferation, but only minor damage of ganglion cells in the beginning. They may disintegrate later on and shrink, especially those in the mamillary bodies, as pointed out by Peters. As the same author mentions, ecto- and meso-dermal proliferation may be, but are not necessarily associated with hemorrhage or with remnants of it in the form of pigment collection. Round cell infiltration may be present, but is not of primary inflammatory character; therefore the term pseudoencephalitis.

The possible findings in *delirium tremens* are by no means characteristic and, if present, consist of non-specific cellular changes with fatty degeneration of elements of the vascular wall. Sometimes we notice liquefaction and granular decay of ganglion cells if death set in following acute aggravation of the basic disease.

Posterior column degeneration and polyneuritis offer more specific findings, but do not belong to our topic.

The majority of authors assumes that death in chronic alcoholism is caused by the following course of events: primary gastro-intestinal damage —liver damage—toxic metabolic products. The latter are supposed to be the cause of the changes in the central nervous system. According to Zillig, liver and brain-stem form a patho-physiological system. Damage in either one of these components may influence the other, a concept already ventilated by E. Pollak many years ago in his studies on glia and liver. For those interested in the subject we suggest study of the papers and books written by Peters, Bender and Schilder, Carmichael and Stern, King and Meekan.

Endogenous psychoses require some introductory remarks. In all these diseases the basic morphopathological processes are still quite unknown. The pathological findings, if any, are often greatly influenced by the chronic clinical course (often for decades) and if patients succumb they do so often from superimposed diseases. In the absence of such pre- or co-existing disease, such morphological findings, if present, and even if characteristic for certain psychoses, are of such a minor degree that their

presence could hardly explain the impressive clinical disturbances or defects in thinking, action, feeling and will power, as seen e.g., in schizophrenia, or in major epileptic seizures. On the other hand, we still have well-founded reason to assume that, especially in schizophrenia, morphological investigations are promising, even more so following the research undertaken by C. and O. Vogt and others, not to speak of electronmicroscopy, which has as yet hardly entered the field of neuropathology.

The different types of epilepsy, especially the true or essential form, are of great forensic interest. Not only may an epileptic die in circumstances indirectly related to his basic disease, like drowning during a fit but, as B. Mueller puts it, he may die following repeated epileptic seizures, (status epilepticus) and such a sudden death may reveal nothing but cerebral edema or swelling. This is of special importance if the deceased was not under the care of an institution when he died, but passed away when alone and was found dead. The autopsy may show gross changes or no changes at all. No matter whether a proper clinical history is available or not, Ammon's horn of the brain and the cerebellum (Spielmeyer) should always be examined routinely and carefully in such a case. Symptomatic epilepsy produced by scars, adhesions and tumors, will not be discussed here as, in most cases, the changes produced by the causative pathology will explain the problem sufficiently.

The anatomical changes in epilepsy, as pointed out by Peters, are consequences of the disease and not its cause. Even more are they the consequence of but one symptom, viz., the tonic and clonic fits. Following Peters and others, the preparoxysmal phase is associated with anemia in different areas of the brain, and more or less extensive hyperemia and increase of the volume of the brain accompany the onset of the spasms. According to Spielmeyer, the partial cerebral anemia is a functional vascular spasm, i.e., a spasm lacking morphological changes in the wall of the contracted vessel. These spasms may or may not lead to complete or incomplete softenings of the brain. The greater hippocampus (Ammon's horn) participates in this process quite often, as evidenced by cellular disintegration and reduction of the number of cells, which later on are substituted by a rather densely arranged glial network. These hippocampal changes may be uni- or bilateral. If death sets in during a paroxysm, the ganglion cells of the hippocampus suffer from ischemia. As already indicated, the cerebellum may also present rather characteristic changes in cases of genuine epilepsy, as proven by severe damage and destruction of the Purkinje cells. This is followed later by gliosis and potential changes in the granular layer (Spielmeyer). These cerebellar changes may produce shrinkage and induration of the involved parts. Patches and streaks brought about by local disintegration as a consequence of ischemia may also be met with in other areas of the brain. If patients survive for a period of time, all these processes are followed by gliosis.

Gruenthal (1947) attributes special importance to the hippocampus with regard to intellect and affect, and discusses one of his cases where, in the course of epilepsy associated with dementia, the hippocampus was destroyed. Spielmeyer contradicted this connexion, because he found quite impressive destruction of the hippocampus to be present in about 80% of cases of epilepsy not associated with dementia.

The changes found in the brains of epileptics are primarily the consequence of hypoxemia caused by pre-paroxysmal vascular disturbances. Still following Peter's discussion on the subject, one has to consider that immature tissue is more susceptible to hypoxemia and therefore the infantile brain is damaged by epileptic fits more easily. This means epileptic children will show more areas of softening than will adults. According to Scholz, there is a rather close casual connexion between epileptic fits and certain types of exogenous idiocy. The pathologist, especially the legal pathologist, has to be aware of the fact that other pathology, like thrombosis or emboli leading to softening may be superimposed on pre-existing epilepsy or may be brought on by other co-existing diseases, like arteriosclerosis. Therefore these changes have nothing to do with the basic epilepsy. As the causes of vascular disturbances in cases of genuine epilepsy are not known, the pathologist is unable to state whether such a cause is of endo- or exogenous origin with reference to the brain. Genuine epilepsy may be assumed only if cerebral changes like arteriosclerosis, traumatic scar tissue, hydrocephalus or primary or secondary tumors are absent. In other words, genuine epilepsy should come into consideration only if symptomatic epilepsy may be excluded. Most probably the fit in genuine epilepsy is nothing but a symptom of a basic (metabolic?) disease, unknown as yet.

Bleuler (in discussing the group of schizophrenia) states: 'Anatomical findings, though often present, do not correspond with the severity of the manifest symptoms, and there are some other elements in favor of the theory that the genesis of this disease is a psychic one . . . aggravations and improvements in the patient's condition are often psychically determined . . . the significance of the undeniable anatomical findings may be questioned on the basis of Schott's interpretation of atrophy as the result of inactivity; or, according to Jung, by regarding them as the consequence of toxins produced by affect. The symptomatology of this disease differs basically from that of any other known organic or toxic disorder . . .'.

However, there are anatomical findings but they are not specific. To evaluate their significance it seems reasonable to restrict this discussion only to fatal cases of schizophrenia, which means to those where the psychosis is *the* cause of death. According to Bleuler, these fatal cases all have a catatonic character. We do not include all those cases which die from poor nutrition, exhaustion from incessant activity, epileptic attacks or infections. The patients we are interested in, die in spite of special feeding methods and special diet and in spite of every possible nursing care, from generalized atrophy, often within a few weeks or months. Following

Bleuler's outline further, we know of a special group of schizophrenic cases which die from a kind of cerebral catatonic paralysis. In the course of events these patients finally develop such a state that not even negative reactions, as in other catatonics, are available. Even blinking of the eyes ceases. Such a situation may lead to autopsy findings quite characteristic of this type of catatonia. We refer to necrotic areas of the cornea, often associated with extensive ulceration.

Many investigations have been performed and are still in process to correlate definite and indefinite pathological lesions with the clinical symptoms in schizophrenia (A. B. Baker). Findings reported by Marchand, Meyer, Elvidge and Reed, Josephy, Watanabe, Wohlfahrt, Marcus, Gjersing, Scheid, Jahn and Greving and many others reveal more or less pertinent lesions in single cases or in groups of cases, but these lesions are not pathognomonic. As Peters points out convincingly, so far we do not know of a somatic syndrome which would fit schizophrenia. The matter becomes more complicated if one realizes that, in general, this psychosis runs a very chronic course, often spreading over decades, and the fatal end in the individual cases is mostly brought on by superimposed pathology, not related to the basic disease. Accordingly, the histological findings (macroscopically the brain of schizophrenia does not show any characteristic changes) vary from one extreme to the other. Prior to the aforementioned authors, it was Alzheimer who, as early as 1900, claimed to have found focal and disseminated disappearance of cells. Many others have since reported similar findings. These foci may become so large that almost the whole cortex is involved. These areas are not displaced by glia, an indication of a slow growing process. Due to the fact that these areas are often widely disseminated, they cannot always be demonstrated, but if found, their situation sometimes corresponds to certain clinical symptoms. Miskolcy goes so far as to claim that optic and acoustic hallucinations may be based on cell-lacking areas in the occipital or temporal regions. Peters was able to demonstrate that similar areas were found in the brain of executed individuals who never suffered from schizophrenia. Spielmeyer, Gellerstedt and many others found that the same changes existed in soldiers killed in action and in persons who died suddenly. The same applies to other manifestations, like cellular sclerosis with vacuolar degeneration described by Fuenfgeld in schizophrenias, which were also found in other diseases. Though all the positive and negative histological findings in schizophrenia are rather contradictory, we cannot omit mention of important investigations by C. and O. Vogt, who found the previously mentioned vacuolar degeneration and cellular sclerosis in the medial nucleus of the thalamic region in catatonics. They describe these cellular changes as associated with premature fatty degeneration, terminating in glial reaction. The areas involved are related to prefrontal regions of the brain. According to C. and O. Vogt, the fatty changes are primary, precede the catatonia and are 'a partial anatomical substrate' of schizophrenia. Neither these

nor the other findings form a sound enough basis to entitle the pathologist to draw far-reaching conclusions, especially with reference to forensic problems. Circulatory disturbances similar to those in epilepsy, and often observed in acute fatal catatonias, are non-specific and are not *the* cause of the psychosis. The same applies to edema of the brain (see above) and the changes in the liver associated with schizophrenia, described by Gaupp. This author found steatosis in the central part of the liver lobules in cases which died in acute catatonic stupor. Sometimes it was associated with areas of fresher or older necrosis in the lobules. Gaupp observed 22 cases of schizophrenia, including 3 cases of acute delirium. In 5 cases he found bile thrombi in the centers of the lobules without destruction of the liver texture. The author attributes all these liver changes to functional disturbances, a consequence of primary epithelial damage. Gaupp assumes toxic substances, derived from the abnormal metabolism present in catatonia. This may hold true as long as other superimposed pathology, which may produce similar liver changes, like aspiration pneumonia, does not interfere (Altmann). Altmann does not deny that liver damage in the course of certain schizophrenias with catatonia may form a link in the chain of so far unknown patho-physiological events.

To-day shock therapy is one of the most frequently used means of treatment of schizophrenia and certain other psychoses. From time to time, fatal accidents occur, during or after the administration of shock therapy. They may carry some forensic importance. After the use of insulin, the brain may show petechiae and areas of softening. Accordingly, hemorrhages are seen under the microscope, associated with focal or diffuse glial proliferation as well as areas of demyelinization and even cyst formation. Metrazol and electric shock are less damaging than insulin; if present, the damage is similar. Prefrontal lobotomy executed in schizophrenia associated with states of anxiety and compulsion, if fatal, or if examined some time after surgery, shows only changes comparable with those seen in animal experiments, after severment of parts of brain.

All histological reports supposed to prove an anatomical basis in manic depressive psychosis have turned out to be erroneous. The same applies to involution psychosis.

Summarizing, we may therefore say that, in spite of numerous reports indicating certain potential histological findings in endogenous psychoses, they are not pathognomonic, though perhaps valid for single cases and promising for the future. Their forensic value lies only in the potential support of the psychiatrist. Only if the latter has diagnosed a certain psychosis during life may morphological findings, if present, support such a diagnosis. Never, however, is the pathologist able to establish the diagnosis of intravital insanity on his findings alone. His position in these cases is contrary to that in surgical procedures. While in the latter he may be of decisive help in making or verifying a diagnosis, in forensic psychiatry the pathologist is dependent on the report of the psychiatrist.

Electroencephalography and Forensic Medicine

II. Forensic Implications

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Clinical Significance of Electroencephalography

There are two main spheres in which electroencephalography is of potential forensic significance. One concerns the extent to which abnormalities or disturbances in brain function may underlie criminal behaviour, or affect responsibility for illegal actions; the other where a plaintiff may seek compensation or legal redress because of an injury to his brain, which may or may not have caused epilepsy. The EEG can be valuable in each of these spheres, since it can provide objective evidence of disturbance in brain function. It is, therefore, advisable, before proceeding to a detailed consideration of the EEG in criminal delinquency, to give a brief survey of the degree of significance of the EEG as an indication of epilepsy or brain damage. Superficially, one might assume that this is straightforward, since it should require little more than a study of the relative incidences of positive EEG abnormalities among adequate samples of relevant clinical groups, these being compared with a normal population. Unfortunately, however, the figures which have been presented in the literature on the subject have often varied widely.

Probable reasons for this are:

1. Differences in diagnostic criteria;
2. Differences in clinical populations studied; and
3. Differences in criteria of normality or abnormality in the EEG.

Positive moves towards rectification of the last group have been going on for several years[28] and the general picture is fortunately much clearer than it was, say ten years ago.

There are various types and grades of abnormality in electroencephalography. While it cannot be said that any of these are diagnostic of a specific brain disease, it is true that some are of greater clinical significance than others. For example, the bilateral 3 per second wave-and-spike is strongly suggestive of *petit mal* epilepsy (although it is not exclusive to

this disorder). Also any focal or paroxysmal discharge is of a high degree of significance. The former is suggestive of a localized, acquired brain disorder, the latter of a disposition to epileptic seizures. Clinical aspects of electroencephalography were briefly discussed[28] in Part I, so that it is sufficient here to present the statistics and conclusions which underlie the assessment of significance of an abnormal EEG. It should be noted that a normal EEG does not exclude the possibility of epilepsy, since the EEG of an epileptic need not always be abnormal. However, it is usually reasonable to assume that if the EEG is normal during a typical "fit", then that person is either hysterical or a malingerer.

Epilepsy. Some idea of the incidence of abnormal EEG's in this disease may be gained from Gibbs[6] who, in a medico-legal paper reported that of 1,260 epileptics there were 38.6% paroxysmal EEG's, 19.8% very abnormal, 28.6% slightly abnormal, and 13% normal. A more recent study by Grey Walter[14] revealed that, of 2,200 'persons complaining of fits', there were 52% abnormal records; 875 of these persons had a firm diagnosis of epilepsy, and among these there were 66% with abnormal EEG's of various types. (This figure agrees well with the present author's finding of 61% abnormal EEG's among 350 epileptics.[25]) After a consideration of distributions of the abnormal EEG's according to a variety of clinical headings, Walter was able to conclude that of every 7 cases with complaint of fits, the EEG would be indispensable for diagnosis in 1, of value as a supplement to clinical methods in 3 or 4, and of no positive value in the remaining 2 or 3, in that the records would be either normal or non-specifically abnormal.

Another point of importance is that the incidence of abnormal EEG's among epileptics varies according to age and kind of epilepsy. For example, Walter found that the percentage abnormality in epileptics as a group declines slowly from about 95% at age 5 to 40% at age 30 and to 10% at 50,[14] whilst Mundy-Castle found abnormality incidences among *grand mal*, *petit mal* and traumatic epilepsy of 48%, 67% and 92% respectively.[25]

These figures give a good idea of the clinical potentialities of the EEG when relatively few activation procedures are included, but (as was pointed out in Part I), the incidence of clinically helpful EEG's can be increased if many such techniques are included, so that these figures should not be looked upon as final.

Suspected Brain Injury. Gibbs[6] has presented a useful list of conclusions regarding the significance of an abnormal EEG in this sphere. Some examples of these are:

'i. Focal EEG abnormality is strongly suggestive of brain damage.

ii. Other things being equal, if generalized EEG abnormality is present 3-4 months after a mild head injury, the chances are 16:1 that the abnormality antedated the injury.

iv. In post-traumatic cases, even though the EEG is normal, the brain may be damaged.'

The interested reader is referred to the original article for the remainder of this factual material.

The finding of Gibbs *et al.*[7] that abnormalities are greatest soon after a head injury and tend to subside in a few days or weeks unless epilepsy develops, is of particular importance in relation to accident cases, when EEG's should be recorded at frequent intervals. It is also possible (as indicated in the Gibbs example (ii) above) that the residual effects of a cerebral trauma may be a generalized rather than a focal EEG abnormality, and as Cobb[14] has pointed out in an excellent review of the EEG in cerebral trauma, this cannot be distinguished in any respect from the so-called 'constitutional dysrhythmia' associated with idiopathic epilepsy. ('Dysrhythmia' describes irregular EEG activity made up of a number of slow and faster components). Hence the importance of Gibbs' statements concerning probabilities. Cobb sums up his review of the problem by stating:

'It is probable that, when records are made two or more years after head injury, a generalized abnormality is of little or no greater significance than it would be in the absence of a history of injury, while a focal abnormality is most probably the result of injury and correlates well with the site of injury and the occurrence of focal fits and residual neurological signs'.

The only further comment to be made is that in recent years there has been an increasing number of reports of a small number of cases with unilateral foci from the *temporal* lobes without any evidence of an underlying lesion, so that a focal EEG abnormality from the temporal region is not quite so strongly suggestive of brain damage as are focal EEG disturbances from other areas of the brain. These temporal lobe focal abnormalities are discussed at length below.

An example of the potential forensic value of the EEG in cases of suspected cerebral trauma is provided by the following case:

Lt. X, an Air Force pilot, was returned from combat duties because his behaviour suggested a lack of moral fibre. Early one morning, before an operational flight, he had felt sick and vomited. He felt giddy while returning from the mission, but turned on the oxygen and felt well. He was sick and vomited again that evening and again the next day. He complained of abdominal pains. After admission to hospital there were several further attacks of abdominal pain with nausea and vomiting, but examinations revealed no evidence of organic disease and no abnormalities. In general, the patient was extremely vague about himself and his complaints. A psychiatric report concluded that the symptoms were of psychogenic origin, and were related to anxiety about future flying, in view of a crash, after take-off, three months earlier. Of this crash, the patient could only remember the engine failing at 500 feet, and that he opened his canopy and started to turn back towards the aerodrome. The next thing he knew was that he was being taken to hospital in an ambulance. He suffered a lacerated wound of his groin and a head injury, but there was no fracture of the skull; the X-ray report was normal; an EEG examination was not performed.

Investigations after his return from operational duties included an EEG examination. This disclosed numerous paroxysmal bursts of theta rhythm localized to the

left temporal area, together with occasional generalized or frontal bursts of theta. Several left temporal sharp waves were also observed. It was concluded that the EEG raised the possibility of an organic disturbance in the left temporal area, with possible involvement of deeper structures. A sample from his EEG is shown in Fig. 1.

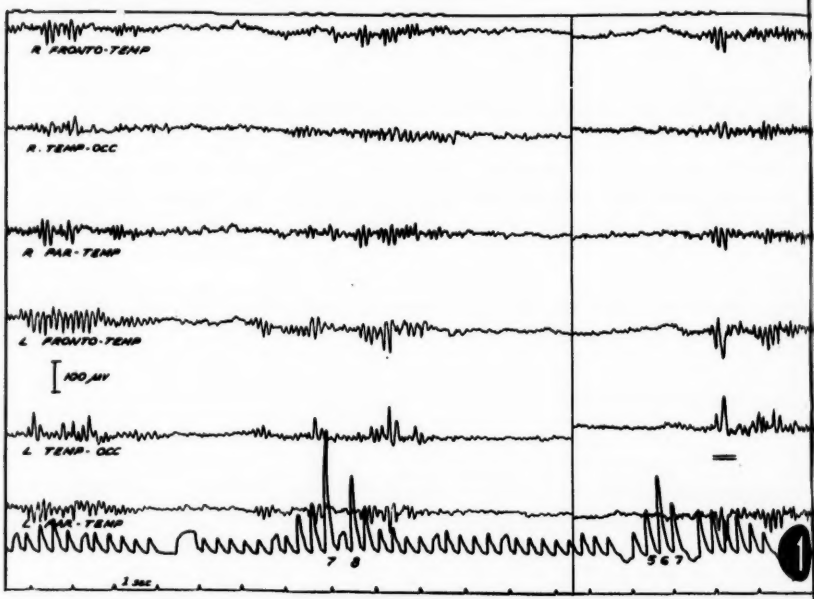

Fig. 1. Two samples from the EEG of the pilot discussed in the text. In both instances, the asymme between the hemispheres is obvious, with disturbances more marked on the left. In "A" (on l there are several sharp-tipped waves from the left temporal region, and the analyser records considera 7 and 8 c./sec. activity as out-of-phase between channels 4 and 5, whose common electrode is over left temporal lobe. In "B" (on right), a very clear focal, sharp-tipped, theta wave is seen arising fr the same area.

The EEG report, taken in conjunction with the head injury, and concussion sustained in his flying accident, and also the permanent loss of memory concerning the crash, led to the conclusion that the patient's symptoms and lack of confidence *may* have had an organic basis referable to the flying accident. These facts were held to be incompatible with malingering.

Electroencephalography and Criminal Behaviour

Nature and Incidence of EEG Abnormality among Delinquents

From 1937 onwards there have been numerous reports of a high incidence of abnormal EEG's among behaviour problem children and adolescents, and among certain adult psychopathic personalities, the commonest type

of abnormality being excessive theta rhythm.[2, 3, 9, 11, 12, 16, 18, 20, 22, 31-34] It is of course true that slow activity is a characteristic feature of the EEG's of normal children until the age of about 5-6 years, and that adult criteria of normality or abnormality in the EEG cannot be applied to children's EEG's; nevertheless the consistent, persisting trend of the above reports is sufficient to make one accept that one of the commonest abnormalities in juvenile delinquency and aggressive psychopathy is excessive slow activity, particularly of theta rhythm. As was discussed in Part I, this perisistence of theta rhythm with increasing age is attributed to delayed or impeded cortical maturation, and it is therefore not surprising that high incidences of such EEG abnormality are found in behaviour deviate groups exhibiting immaturity. For example, Michaels and Secunda[24] found that enuresis, which they considered to be a reflection of immaturity, occurred more commonly than any other neurotic trait among 122 children, and that it was positively associated with abnormality in the EEG. This finding was later confirmed by Michaels[23] who found that of other individual traits, only fire-setting showed a positive association with EEG abnormality. He wisely observes that it would be dangerous to accept too readily the conclusion that there is a 1:1 correlation between EEG abnormality and psychopathy. In this connexion, Hill[14] has stated that the general finding of excess slow activity amongst problem children would appear to be better correlated with hyperkinesis, irritability, temper tantrums and enuresis than with such a complex syndrome as delinquency. It is, however, important to note that Gallagher *et al.*[5] reported a correlation between 'poor' personality and abnormality in the EEG among normal adolescents aged 14-15 years. Also Kennard, in a comprehensive review of studies of the EEG in psychological disorders, reports that 'good' personality and 'good' EEG have been related in many ways.[19]

The determining factors concerning the disappearance of theta rhythm with age are not fully understood; no doubt both genetic and environmental factors are important. It is true, however, that with increasing age slow activity tends to disappear from the EEG's of both normal and abnormal children, and this kind of abnormality, together with psychopathic behaviour, occurs less and less with older adults; these facts are best explained in terms of the operation of a maturation process.

EEG in Juvenile Delinqnency

An interesting extension of electroencephalography has recently been made by Hodge *et al.*[17] These workers undertook a comprehensive study of juvenile delinquency, the results of which may be of considerable forensic importance.

The investigation involved a detailed electroencephalographic and psychosocial appraisal of 100 young male delinquents aged from 10 to 17 years.

The most surprising outcome of the investigation was that 84% of the EEG's were abnormal, whereas only 5% of the EEG's from a non-delinquent juvenile control group were abnormal by the same standards. It must be noted, however, that only 5 of the abnormal EEG's of the delinquent group were in any sense indicative of a clinical ailment, so that the bulk of these abnormalities must be regarded as statistical rather than pathological in their meaning at the time of classification. All of the latter abnormal EEG's contained theta rhythms, while 68 contained low voltage, poorly localized *delta* rhythms. The former was expected, in view of the well-established relationship between theta rhythm and immaturity, but the incidence of delta rhythm was far higher than had been anticipated.

The incidence of abnormal EEG's among adult psychopathic personalities is higher than among psychoneurotic or psychotic groups, excepting those with organic brain disease.[9, 12, 14] The reported percentages vary from about 50-75%, probably due to differences in the composition of groups studied and in diagnostic criteria. As already noted, the greatest incidence is found amongst aggressive psychopaths, where the characteristic abnormality is excessive post-central theta rhythm, probably of deep origin. Hill[9] has also reported that a number of these aggressive psychopaths possessed a unilateral, often right-sided, temporal lobe focus of slow activity (3-5 c./sec.), whilst bilateral temporal lobe foci were common. In advancing his hypothesis that the aggressive psychopath with this type of EEG abnormality is an individual in whom cortical maturation has been impeded or retarded, Hill draws on the finding that these people, like children, are particularly liable to show high-voltage 2-3 c./sec. waves during or after hyperventilation,[12] thereby suggesting the possibility of a defective acid-base balance control. He suggests that this is also related to a maturation process: such a response in children is of no clinical significance, and disappears with advancing age.

These theories of Hill are amply supported by a recent study by Stafford-Clark *et al.*,[36] who found a significant correlation between EEG abnormality and aggressive psychopathy, both including and excluding epileptics and organic cases. They also found that, at all ages, responses to hyperventilation were more abnormal in psychopaths than in controls, and that abnormal hyperventilation responses were rare over 30 years. The type of abnormal response was excessive theta rhythm, chiefly from central areas, this being regarded as suggestive of physiological immaturity. The authors incorporated several other approaches to assist in their study of the psychopath and, on the basis of all these, concluded that the fundamental abnormality in such persons is a failure in maturation. The evidence in general therefore supports the view that the EEG can provide a valuable indication of the level of cortical maturation.

So far as the general criminal population is concerned, a large number of studies has been undertaken, often with rather conflicting findings. The

reported incidence of abnormal EEG's among criminals is, however, usually greater than among normal subjects, although one does come across such opposed reports as that of Gibbs *et al.*,[8] who found no significant difference in EEG abnormality between a 'normal' prisoner population and the general population; and the more recent statement of Stafford-Clark *et al.*[36]: 'Even the normal criminal shows a surprisingly high incidence of EEG abnormalities'. The probable reasons for these discrepancies are, as usual, differences in EEG criteria and in the groups under study, and also differences in age. For example, Hill[14] suggests that the surprising finding by Gibbs *et al.*[8] of no correlation between EEG abnormality and aggressive psychopathy would have been different if the 79 murderers who were included in that study had been examined at the time of their crimes, since there is little doubt that the incidence of psychopathic and epileptic EEG abnormalities diminishes with age. In support of this suggestion is a recent report of Levy and Kennard,[21] who found that in a group of 100 convicts, there was a positive correlation between abnormal EEG's, age at time of EEG test, and age at time of first offence. They also found that their prison population had a greater incidence of abnormal EEG's (30%) than the normal population, accepting this as confirmation of a general finding. Oddly, they found no difference in incidence of EEG abnormality between crimes of violence and non-violence, but do state that more detailed study and observation might have produced some relationship between paroxysmal behaviour and paroxysmal EEG records.

Studies of Murderers and Aggressive Psychopaths

An important distinction which should be stressed here is that between *maturational* and *'epileptic'* abnormalities in the EEG. The former are non-paroxysmal, the latter paroxysmal, Hill[11] having suggested that they be called *homeostatic* in the sense that they are related to recovery processes from illness rather than to the illnesses themselves (see also Roth[30]). Thus, given suitable or sufficient stimulation, any person, including normal subjects, can be induced to display the paroxysmal EEG activity which is associated with an epileptic seizure. It is the *threshold* of the seizure which tends to be lower in epilepsy and certain other pathological conditions. The biological significance of the maturational abnormalities has already been discussed, in that they relate to defective maturation in the central nervous system. It should also be made clear that these two classes of abnormality are far from being mutually exclusive; in fact they are often found together. Furthermore, both are also always found in a small percentage of any large group of subjects.

The importance of this distinction will be made clear in the course of the following discussion, particularly in relation to epilepsy and aggression, where careful examination of known facts negates any unique or peculiar relationship between the two.

Perhaps the first significant indicator of the forensic possibilities of electro-encephalography in murder cases is a report by Hill and Sargant [13] in 1943. In this case, the accused killed his mother by stabbing with a kitchen knife. He stated that he had quarrelled with his mother, saw a kitchen knife and 'something came over him' so that he was like a 'homicidal maniac'.

EEG examinations revealed that whenever his blood sugar was below a critical level, the EEG was abnormal and hyperventilation would induce an abnormal response, this being associated with some degree of impairment of judgment and clouding of consciousness. The history indicated almost certainly that at the time of the killing his blood sugar level was below the critical level, and that hyperventilation had occurred. It was concluded that although from the legal point of view the accused knew what he was doing at the time when he killed his mother, and knew that what he was doing was wrong, at that time his brain was functioning abnormally, so as to impair his judgment and render him unable to appreciate fully the nature of the act. The prisoner was pronounced 'guilty but insane'.

This is an excellent example of the use to which electro-encephalography can be put in such cases. It should be noted that very careful and detailed methods were taken to reproduce in the laboratory as exactly as possible a state in the accused as was comparable with that at the time of the crime. This involved, among other things, causing the accused to drink the same amount of mild ale as on that night, thereby enabling a fairly accurate estimation of his blood sugar level at the time of the crime. From the legal point of view, it would always be important to ensure, in comparable cases, that the EEG methodology allows for all possible extraneous factors and, when necessary, incorporates appropriate activation procedures other than those used routinely.

General Findings Derived from Studies of Groups of Murderers. It must be borne in mind that conclusions based on these studies are primarily referable to the group studied, and not necessarily to any individual case. The first comprehensive study of a large group of murder cases is that reported by Stafford-Clark and Taylor [35] in 1949. Since a later paper by Hill and Pond [15] is in many respects an extension of this study, the two will be considered together. A total of 105 cases was investigated, the first-named authors reporting on the first 64 cases, Hill and Pond on the whole group. Crimes were sub-divided into the following five categories:

1. Killing incidental to another crime or in self-defence.
2. Killing clearly motivated, or resulting from violence during another crime.
3. Killing which is apparently motiveless, or with very slight motive.
4. Killing in which some kind of sexual activity occurred the the time of the murder.
5. Killings by insane persons who were either unfit to plead or were found insane at trial or later at a statutory enquiry.

Both studies revealed a persisting characteristic association concerning

the incidence of abnormal EEG's among these sub-groups, viz. that predominantly normal EEG's were found in Groups 1 and 2, predominantly abnormal EEG's in Groups 3 and 5. The figures for Group 4 were too small for meaningful comparison with the remaining groups, although the incidence of abnormal EEG's in this group (5 out of 9) is clearly considerably greater than in a normal population.

Stafford-Clark and Taylor found that in Group 1, which they call the 'accidental murder' group, the incidence of abnormal EEG's was less than 10% (actually 1 : 10), which they consider to be comparable to that in a normal population, whilst Group 2 (the motivated group) contained 25% abnormal EEG's. They compare the latter incidence with that of the prison population of America.

However, although Hill and Pond (in their study of the extended group of murderers) claim that their findings only underline those of Stafford-Clark and Taylor, inspection of their figures shows that the respective percentages of abnormal EEG's in Groups 1 and 2 are 23.5% and 21.5%. In other words, the difference between these two groups has not been maintained and, in fact, both incidences appear rather to approach that of the American prison population—a conclusion which is not surprising in view of the figures for criminals mentioned earlier in this paper.

This observation also underlines the danger of deriving conclusions from small samples without resorting to appropriate statistical techniques: a null hypothesis test of the difference in abnormality incidence between Groups 1 and 2 of Stafford-Clark and Taylor reveals that it is not significant, the probability of its occurring by chance being greater than 25%. (The usual criterion for significance is 5% or lower.) A similar test applied to the figures given by Hill and Pond for Groups 1 and 2 reveals the difference to be totally insignificant, the probability of its occurring by chance being well over 50%. These statistical tests therefore confirm that Groups 1 and 2 can be drawn from one and the same EEG population. This population appears to be closer to the prison than to the general population, in terms of abnormality incidence.

One of the most important observations from these murderer studies is the very high incidence of abnormal EEG's in Group 3, the motiveless or nearly motiveless category, the actual figure being 78%. This is slightly above that for the insane group (67.5%). The over-all incidence of abnormality among the murderers was 47.5%, whilst there is a clear decrease in percentage abnormality with increasing age. Hill and Pond state that there is a slight increase in percentage abnormality over 40 years, but although this fits in with the advent of cerebral degenerative disorders and accords with data from studies of psychiatric material, the statement should be treated with reserve, since their tabular material shows that only in the 41-45 year age group is there a noticeable increase in percentage abnor-

mality, whilst the total number of their subjects over 40 is too small for the purported increase not to be attributable to chance.

These two studies reveal quite clearly that the murderer population is electro-encephalographically abnormal. Another important observation is that the incidence of epileptics among these murderers is at least 32 times as great as is found in the general population. This is definite evidence of a relationship between murder and epilepsy in some murderers, but Hill and Pond stress that this does not necessarily mean that such murders are committed in epileptic seizures or in post-epileptic automatisms. Indeed, they report that most of the available evidence is against this, and that violent or dangerous behaviour is rarely seen during epileptic or post-epileptic automatisms in any patient.

In discussing this problem they point out the forensic importance of knowing the conditions under which dangerous behaviour is witnessed in relation to a fit. The purposive, co-ordinated and sustained behaviour necessary for a spontaneous assault could not possibly occur during the cortical discharge of a major or minor epileptic seizure. Recovery from the latter is immediate and without automatism, but after a *grand mal* attack there is a variable period of mental confusion, accompanied by profound disturbances in the electrical rhythms of the brain, and Hill and Pond agree that undoubtedly some murders have been committed during this period, although none of their own series was in this category.

Antisocial behaviour in an epileptic is, however, usually ascribed to the type of seizure known as psychomotor, since this may involve a partial hold on consciousness during the actual cerebral epileptic discharge. Hill[10] reports that in a dozen or so cases in which EEG's were recorded during these attacks, there was slow activity from one side of the brain, accompanied by semi-purposive movements such as fumbling and chewing, but behaviour was never so sufficiently co-ordinated as to constitute an aggressive threat.

The general conclusion derived from these observations is that the aggressive acts of epileptics are not narrowly related to their actual fits (defined, after Hughlings Jackson, as the clinical correlate of a sudden excessive discharge of grey matter), but that there must instead be some more general relationship.

A clue to one common factor in such relationship is provided by the fact that of all epilepsies it is the psychomotor category which is most commonly associated with behaviour disturbances. As already indicated, the epileptic focus in this disorder is located in the temporal lobe, and Rey, *et al.*[29] have shown that even non-epileptic temporal lobe foci are particularly liable to be associated with behaviour disorders. They report the following features as common with such foci: defective personality in the form of an inability to adapt to environment or to social life; occupational instability; irresponsibility; immaturity; over-dependence; impulsiveness;

nervousness; egocentricity; irritability; aggressiveness. They also state that it is always difficult to classify these patients according to any of the accepted diagnostic criteria, even if psychopathy is included. These disturbances in behaviour are attributed to incomplete temporal lobe maturation, the temporal lobe being regarded as the probable centre of highest integration of cortical activity. In the words of the authors: 'They are children in their EEG's and in their personalities, though chronologically grown up'. As Hill and Pond [15] observe, the temporal lobe also appears to be a high level centre for cortical representation of autonomic function, so that it is probable that these temporal lobe abnormalities may cause autonomic (and consequently emotional) disturbances. The significant factor behind the behaviour of such epileptics would therefore be the temporal lobe maturation defect, and it would be this rather than the epilepsy itself which gives rise to antisocial or aggressive behaviour.

Other factors which may contribute to the relationship between murder and epilepsy are:

i. The epilepsy may be consequent on brain damage, which may itself be sufficient to account for a behaviour disturbance, without reference to the epilepsy.

ii. Periodic fits (which comprise a normal response given sufficient provocation) may be part of some more general cyclic biochemical or physiological changes; and

iii. The homeostatic mechanisms in epilepsy appear to be less efficient than normal.

The last of these explanations is linked with the fact that many aggressive criminal acts are committed under physiological stress, e.g. alcoholic intoxication, hydration, starvation, severe fatigue, etc., so that taking account of all available evidence, it seems justifiable to conclude, as do Hill and Pond[15], that an abnormal EEG may be a latent factor of disturbed cerebral functioning, which can be potentiated by some kind of stress (as occurred in the case of matricide already discussed). In this connexion, the report of Mundy-Castle [26, 27] of a relationship between abnormal responses to flicker and behaviour deviation in children, both epileptic and non-epileptic, may well be relevant, whilst the argument in general would be in accord with the observation of Alström,[1] in his defence of the epileptic, that many of the acts of violence or criminal behaviour committed by epileptics are closely connected with the abuse of alcohol.

This fact that both epileptic and maturational EEG abnormalities are associated with an increased tendency to instability during stress is mentioned by Hill and Pond [15] as one of the factors which narrow the *apparent* difference between these two classes of abnormality; another is the already-mentioned fact that there is no evidence that aggression has any peculiar relation to epilepsy.

The following salient points summarize this rather complex material relating to epilepsy, aggressive behaviour and EEG abnormalities:

1. Aggressive psychopathic disturbances are often accompanied by EEG maturation defects in the form of:

(a) Abnormal theta rhythm, particularly from the temporal areas;
(b) Slow alpha variants; and
(c) Unilateral temporal lobe slow wave foci.

The abnormal behaviour of such persons can be understood in terms of a physiological immaturity in central nervous development.

2. Murderers form an abnormal EEG population, and comprise an unusually high percentage of epileptics. Abnormal EEG's predominate among insane murderers and those whose crimes are unmotivated, or nearly so. There is a high incidence of both epileptic and maturational EEG abnormalities.

3. Violent or dangerous behaviour rarely occurs during epileptic or post-epileptic automatisms, suggesting that the aggressive acts of epileptics are not narrowly related to their actual fits, but that there must be some more general relationship.

4. Several factors may contribute to this relationship, as follows:

(a) Psychomotor epilepsy is the kind most commonly associated with criminal behaviour, and the characteristic EEG abnormality of this disorder is a discharge localized in the temporal lobe. Such a focus is suggestive of incomplete high level cortical integration, and may lead to autonomic disturbances. Thus non-epileptic temporal lobe foci are commonly associated with behaviour disorders, as mentioned in (1) above.

(b) Epilepsy may be the result of brain damage, which could itself account for aggressive behaviour.

(c) The epileptic fits may be part of some more general cyclic changes in the organism.

(d) Both epileptic and maturational EEG abnormalities are associated with an increased tendency to instability during stress. Many of the crimes under discussion are committed under physiological and/or psychological stress, which would account for disturbances in cerebral function at these times.

Further support for some of these observations has recently been provided from a study undertaken in our own laboratory, in which a group of 20 murder and 2 attempted murder cases was investigated. The details of this study have yet to be published, but in the meantime there are several findings which are fully relevant to the present discussion and which should therefore be mentioned here.

Four of the subjects were found 'mentally disordered or defective' before arraignment, 12 on or after arraignment, 3 'guilty but insane' and one committed his crime whilst in the mental hospital. Although only the last 4 of these are cases of proven guilt, in the remaining 16 there is a strong presumption of guilt. (In South African legal procedure the verdict 'guilty but insane' is relatively rarely brought in as compared with British procedure, so that this lack of *proven* guilt is largely a consequence of legal procedure.) Of the remaining subjects, one (an attempted murder case) after being found not mentally defective or disordered during a period of 32 days' observation, pleaded guilty to the charge and was fined £75 or 3 months' imprisonment with compulsory labour; the other was found guilty of culpable homicide and sentenced to 1 year's imprisonment suspended for 3 years.

The following are the important results of the study:

All but 2 of the crimes were either motiveless or with very slight motive,

or motivated by irrational ideas related to a disturbed mental state. The exceptions were the attempted murder and culpable homicide cases just mentioned. Of these, in the former there were the strongest extenuating circumstances together with considerable mental strain and extreme provocation, whilst in the latter the killing was in self-defence during an emotionally disturbed mental state.

Six (27%) of the subjects were confirmed epileptics and 16 (73%) had abnormal EEG's. Of the abnormal EEG's 13 were severely abnormal, including 5 with unilateral temporal lobe foci. One subject with a normal waking EEG was elsewhere shown to have a unilateral temporal lobe spike focus during sleep, making a total of 6 temporal lobe foci in all (27%). Ten of the EEG's (including 4 with temporal lobe foci) showed epileptic abnormalities. Eleven (50%) had abnormal theta rhythm. Examples of the EEG abnormalities found in this study are shown in the samples in the Appendix (Figs. A-H) together with descriptions of the type of crime committed and the associated case-histories.

These results support and strengthen the conclusions of the investigations already discussed. Of particular interest are:

(a) The high incidence of epileptics;
(b) The high incidence of epileptic EEG abnormalities; and
(c) The remarkably high incidence of temporal lobe foci.

It should be added that 3 other EEG's showed asymmetries of bilateral temporal lobe abnormalities, whilst a further 3 showed localized abnormalities or shifting foci suggestive of organic cerebral pathology, one of these being related to premature senility. On the EEG evidence alone therefore, it would seem to be quite reasonable that this is a group in which culpability for criminal or aggressive behaviour should be diminished, in view of the strong probability of abnormalities in cerebral functioning, especially if any unusual stresses prevailed at the time of such crimes. The validity of such reasoning is, of course, substantiated by the conclusions of the Courts regarding the members of this group.

The discussion so far has centred on the problem of epilepsy, psychopathy (in the sense of physiological immaturity) and related EEG abnormalities. It should not be forgotten that some of the EEG abnormalities among e.g. murderers, will be related to developmental or acquired defects in the central nervous system, which may be unrelated to epilepsy or psychopathy. There were 13 such cases in the Hill and Pond study and, as they point out, social custom and the law have long recognized physiological factors as mitigating personal responsibility for antisocial behaviour. These 13 comprised subjects whose personal histories and clinical states indicated a great loading of morbidity of all types, suggesting that the EEG findings are a reflection of these handicaps.

It should also be noted that with the functional psychoses, viz. schizophrenia, manic-depressive psychosis, involutional melancholia and paranoia.

the EEG is as yet still no more than one of the many research tools engaged in investigation of possible physical or physiological factors in these diseases, and discussions of underlying reasons for criminal behaviour by such persons is best made in psychopathological terms.

Possibility of Criminal Differentiation According to EEG and Personality

Another investigation which should be discussed here is that of Levy and Kennard[21], who studied personality and EEG factors in a group of 100 prisoners, and have indicated that it may be possible to distinguish two groups of criminal according to their EEG and personality structure. One of these groups has already been discussed, viz. that comprising EEG maturation defects and instability of EEG during stress. The other group proposed by Levy and Kennard comprises chiefly normal EEG's with a predominance of pure alpha activity (Fig. 2), this kind of record often having been reported as associated with a passive receptive personality. These authors found that whereas the former group showed mainly normal or neurotic profiles on the Minnesota Multiphasic Personality Inventory (M.M.P.I.), the latter tended to show a high 'psychopathic deviate' (P.d.)

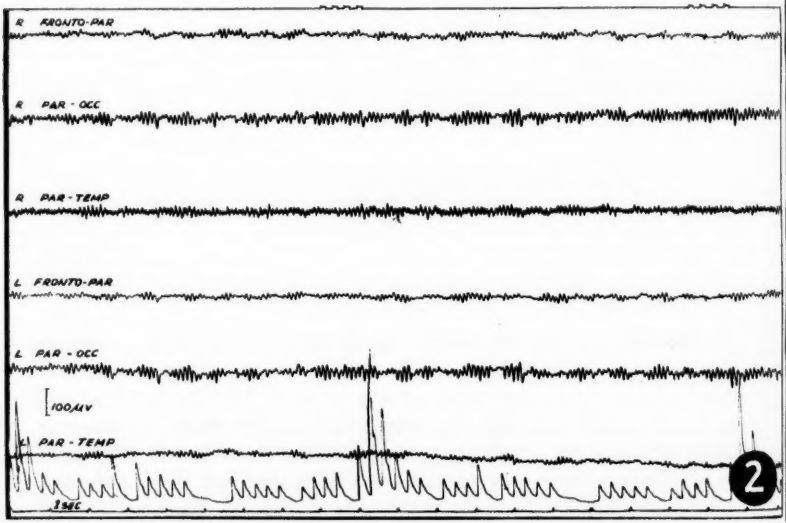

Fig. 2. *Pure Alpha Record.* A male European aged 30. Killed a man in a bar by shooting him thrice. He was intoxicated at the time of the crime, but not drunk. Before committing the crime, he assaulted another man in the bar without any motive. He claimed he had no recollection of any of these events.

Conclusion: pathological intoxication.

The EEG is dominated by continuous, fairly wide-spread medium-high voltage 8—10 c./sec. alpha rhythms, with a clear, consistent peak at 9 c./sec. Discussion of the possible significance of this type of EEG among criminals is presented in the text.

score on this test. They maintain that this 'normal EEG' group possess a fixed personality pattern and would thus require permanent maximal custodial care, whereas the 'EEG defect' group, whose records are compatible with an underlying instability of personality, deserve careful therapy, both somatic and psychological, directed at their specific underlying pathology.

This proposed differentiation is of considerable interest and potential importance, but although their conclusion concerning the 'EEG defect' group is entirely reasonable, that concerning the 'normal EEG' group has less to substantiate it. For example, it has yet to be established whether a high P.d. score on the M.M.P.I. and a 'pure alpha' EEG do in fact reflect a personality pattern which would not be amenable to some form of re-education. Also, they give no test of the statistical significance of the implied relationship between high P.d. scores and such pure alpha records, so that as it stands it cannot be regarded as more than a possible trend, a point which the authors readily admit. Nevertheless, it is true that on EEG grounds alone there is sufficient evidence to differentiate at least two groups of criminal, viz. psychopath with normal EEG and psychopath with EEG maturation defect. It is also relevant that Cohn[4] has claimed that certain patients exhibiting behaviour disorders involving psychopathic traits show fairly continuous relatively high voltage regular alpha rhythms. He suggests that the remarkable stability and lack of fluctuation in amplitude of these rhythms may reflect an inability of the individual to respond to his environment in an adequate way. This observation appears to fit in with the suggestion of Levy and Kennard, and it is clear that further research is needed along these lines.

Summary

An attempt is made to present a comprehensive picture of those aspects of electro-encephalography which are actually or potentially important in forensic medicine. Part I presents a brief history, followed by definitions, descriptions, distinctive characteristics and developmental aspects of the main rhythms encountered in the EEG. Details of the principles underlying electro-encephalography are also presented, together with its methodology, and the training and qualification of electro-encephalographers.

There are two main spheres in which the EEG is of forensic importance —one in which it can be used to verify brain damage or epilepsy consequent on injury, the other where illegal behaviour is related to disturbance in brain function. In Part II this differentiation is used as the background for a presentation of the incidence, nature and significance of EEG abnormalities in epileptic and traumatic states and in various forms of criminal delinquency. Concerning the latter, stress is laid on the differentiation (but not mutual exclusion) between epileptic and maturational EEG abnormalities. The former are paroxysmal, the latter non-paroxysmal. Maturational abnormalities are particularly common in the EEG's of behaviour problem children and aggressive psychopaths. It is also noted that in general there is a

tendency for criminal populations to possess greater percentages of abnormal EEG's than the normal population, but that these percentages decrease with age.

Emphasis is directed to studies of the EEG in murderer populations. The forensic use of the EEG in an individual case is illustrated, followed by an examination of available data on the EEG in murderers. This reveals that murderers form an abnormal EEG population with a high incidence of both epileptic and maturational EEG abnormalities, and comprise an unusually high percentage of epileptics. It is shown, however, that aggressive behaviour in epileptics is not referable strictly to the epilepsy itself, but to several other factors, in particular temporal lobe instability and an increased tendency to central nervous instability during stress, both of these factors also being prominent with maturational EEG abnormalities.

Samples of EEG abnormalities from murderers are displayed, and mention is made of other mechanisms which may operate to diminish culpability in such aggressive behaviour.

The author is particularly indebted to Dr. L. A. Hurst, Physician Superintendent of Sterkfontein Hospital and Senior Lecturer in Psychiatry at the University of the Witwatersrand, for his generous co-operaion and advice concerning the study of murder and attempted murder cases, and is grateful also to Drs. P. Morgan, M. Merlin and P. Booysen of his staff, for their collaboration.

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The author also records his gratitude to the Surgeon-General of the South African Air Force for his helpful co-operation concerning the case of the Air Force pilot.

Appendix

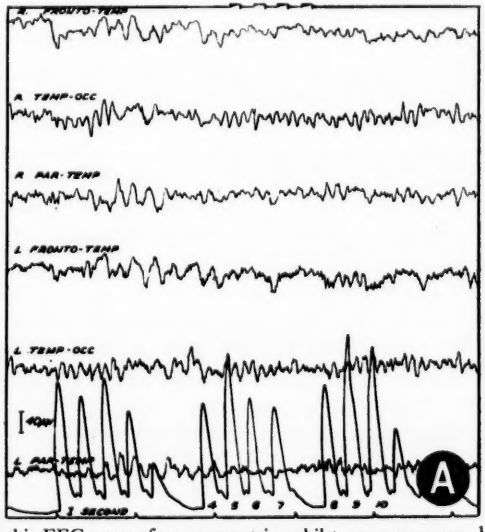


Fig. A. A male African aged 20; catatonic schizophrenia. In 1952 he attempted to rape a small girl and was placed in a mental hospital. In 1954 he murdered a fellow-patient by kicking him on the head with his bare feet, the victim being asleep at the time of the assault. This subject is regarded as often impulsive, violent and dangerous. At the time of the EEG test, he was disorientated, hallucinated and asocial, laughing and talking to himself. During the test he became anxious and uncooperative.

This EEG sample shows diffuse medium voltage theta rhythm, chiefly at 4—5 c./sec., together with lower voltage 8—10 c./sec. alpha rhythms and traces of delta rhythm (but some of the delta recorded by the analyser is due to eye movement). The theta rhythms in this EEG were often asymmetric, whilst some paroxysmal slow disturbances (including delta) were also present, occasionally being large and longer from the left temporal area.

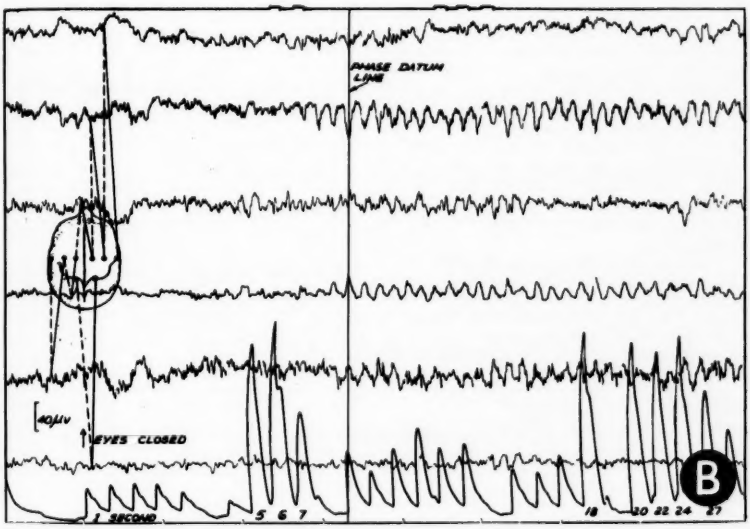


Fig. B. *Excessive Central Theta Rhythm.* A male European aged 35; epileptic psychosis with typical *grand mal* and *petit mal* seizures. Charged with attempted murder. His employer stated that he always made a nuisance of himself, that there was always trouble between him and other men, and that he was always shouting. A witness stated that the accused told him that he suffered from fits and was unable to control himself when angered. The accused also mentioned that he had killed a Native, and produced a long pocket knife, stating that he would use it on anybody who angered him.

On the date of the crime, the accused suddenly opened fire on some of his workmates as they were having lunch, wounding one in the stomach and one in the leg. He stated afterwards that he got cross and shot the men.

The EEG sample shows excessive theta rhythm from the central regions. Clear phase-reversals are present between channels 2 and 4, whilst the frequency analyser indicates out-of-phase activity at 5 and 6 c./sec. between channels 2 and 3, thereby suggesting that the projection of the theta rhythm is sometimes slightly to the right of the midline. There is also excessive fast activity in this EEG (although some of the fast activity in this sample is of muscular origin), again from the central regions, being clearest in channel 3. Occasional generalized paroxysmal bursts of theta rhythm were present in other parts of the EEG.

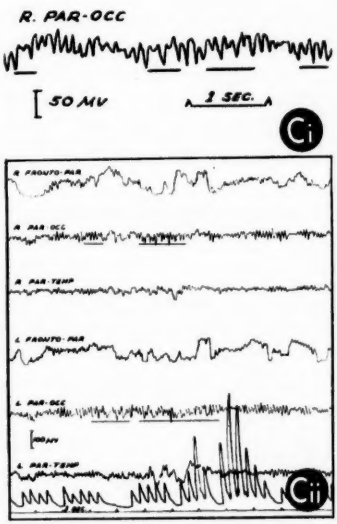


Fig. C. *Slow Alpha Variants.* A male European aged 43. History of psycho-neurotic disturbances, involving obsessive-compulsive behaviour and depression, from 1936 to 1951, when a bilateral frontal leucotomy caused immediate relief. He then became elated and free from worry. In 1952 he had his first epileptic fit (*grand mal*) and since then has had about 10 fits. In 1954 he physically assaulted and killed a woman friend after an argument.

i. Enlarged section of EEG from right parieto-occipital derivation, showing alpha rhythm at 10 c./sec. intermittently disrupted by slow alpha variants at 5 c./sec. These have been underlined.

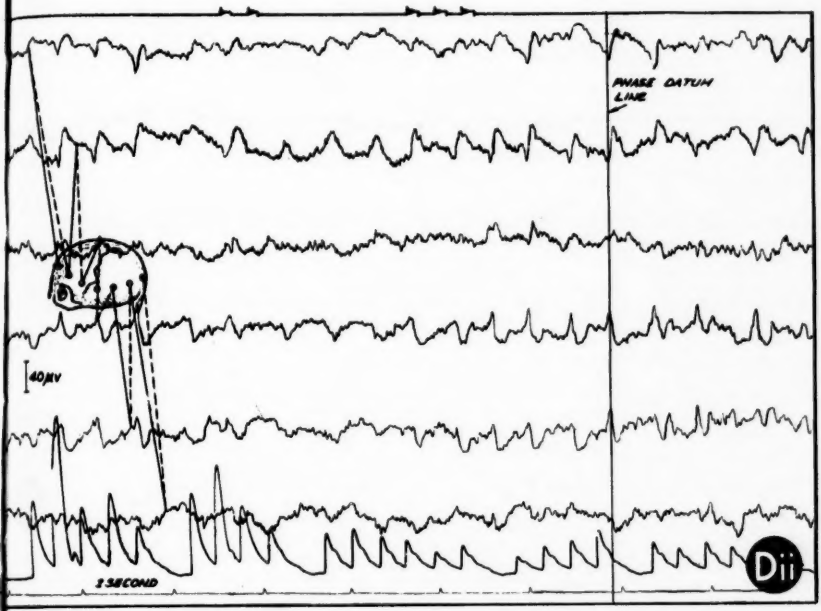
ii. Sample showing all derivations. This EEG contained several other abnormal features besides the alpha variants, viz. excessive theta rhythm; numerous left parieto-temporal slow disturbances at 2—5 c./sec.; occasional paroxysmal disturbances; occasional focal left temporal sharp waves during sleep.

This sample shows considerable asymmetric slow alpha variant and theta activity (underlined) and some irregular delta waves from the left parieto-temporal derivation (not due to movement, as are many of the slow disturbances from the fronto-parietal leads).

Fig. D. *Temporal Lobe Focus—Method of Location.* A male African aged 31; epileptic psychosis with typ*grand mal* seizures. He killed another African male. No details are available concerning the crime. T subject is dull, retarded, disorientated, confused, with vague delusions of persecution.

Di. Routine examination reveals a clear focus of saw-toothed waves recurring at 2 per sec. from left temporal area.

Note isolated focal spike towards end of sample.



ii. Antero-posterior electrode chain shows clear phase-reversals between channels 2 and 4, suggesting at the focus lies between the electrodes of channel 3, i.e. in the anterior temporal lobe. Note that the ave forms are often sharp-tipped and spikelike, also that amplification is greater than in Di.

Diii. Electrodes in a vertical chain from the top of the head to a point below and slightly in front of the ear. Clear phase-reversals between channels 4 and 5, thereby establishing the focus as low in the anterior temporal lobe. Note that amplification is even greater than in Dii.

Div. Electrodes and amplification as in Diii, but paper speed at 6 cm. per second. High paper speeds such as this enable exact and accurate observation of phase relationships. This sample shows clear phase reversals between channels 4 and 5.

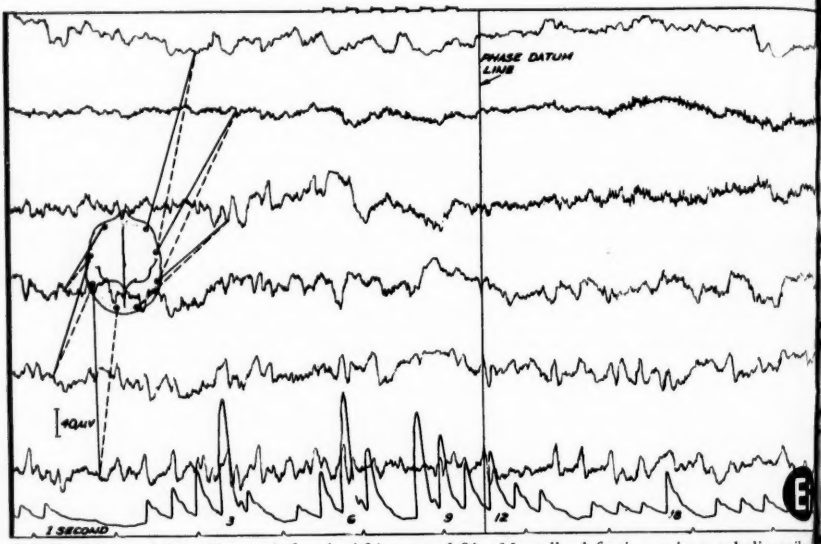


Fig. E. *Temporal Lobe Focus.* A female African aged 21. Mentally defective, microcephalic epilep excited and aggressive; auditory hallucinations; she has *grand mal* attacks with marked post-seiz confusion.

She murdered a newborn child by smashing the skull—the whole occiput was smashed in so tha looked like a mass of soft pliable tissue.

Sample in *Fig.* Ei shows a clear focus of delta and theta activity from the left temporal lobe, toget with several fairly sharp waves. Of interest are the harmonic relationships shown by the analyser, miniscent of those pertaining to alpha variants.

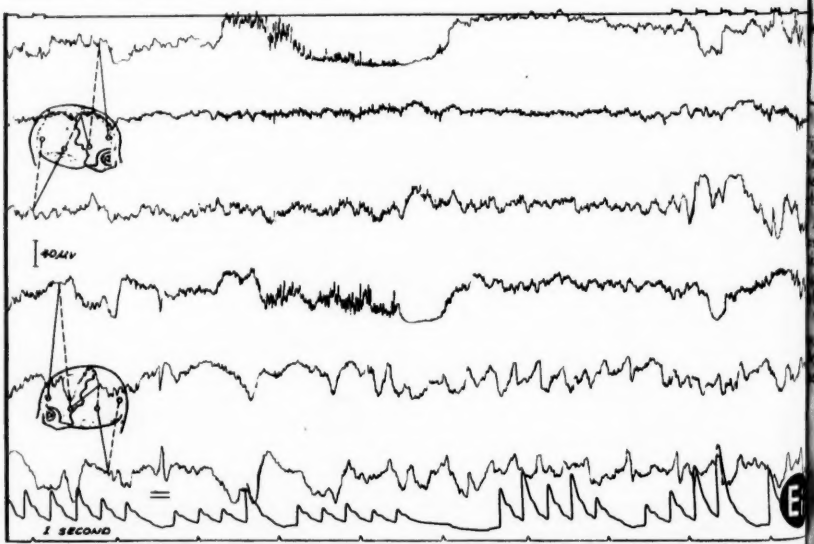
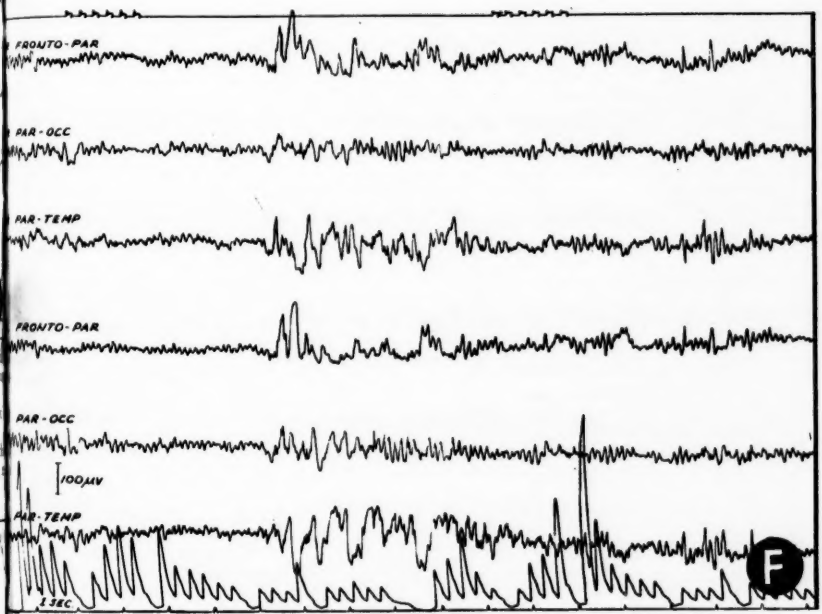


Fig. Eii shows a true spike focus from the same subject. Note that in this example the phase revers between channels 5 and 6, indicating a more posterior origin within the temporal lobe than for abnormalities in *Fig.* Ei, where phase reversals predominate between channels 4 and 5.



g. F. *Paroxysmal Slow Activity.* A female African aged 29. On admission to hospital (25 October 1950) e was dull and disorientated, with auditory hallucinations. A history of epilepsy since childhood *tit mal* and *grand mal*), with an aura of a strange feeling from the stomach, moving up to the chest, ile hearing voices and seeing strange people's faces. Often violent and confused after a seizure.

At the time of the EEG examination (16 February 1954) the patient showed no sign of psychosis and r general behaviour was exemplary. Seizures are diminishing in frequency and there is now no post-zure confusion. Originally committed to a mental hospital after killing her 3-month-old child by throw-g it into a fire. She could only remember running away with the child, and did not know what occurred bsequently.

The EEG sample shows a high voltage bilateral paroxysmal outburst of slow activity, chiefly delta, rticularly prominent from the parieto-temporal derivations, where voltages are greater on the left. te wide-spread medium-high voltage alpha rhythms at 8—9 c./sec. often disrupted by underlying eta rhythm or by isolated sharp waves.

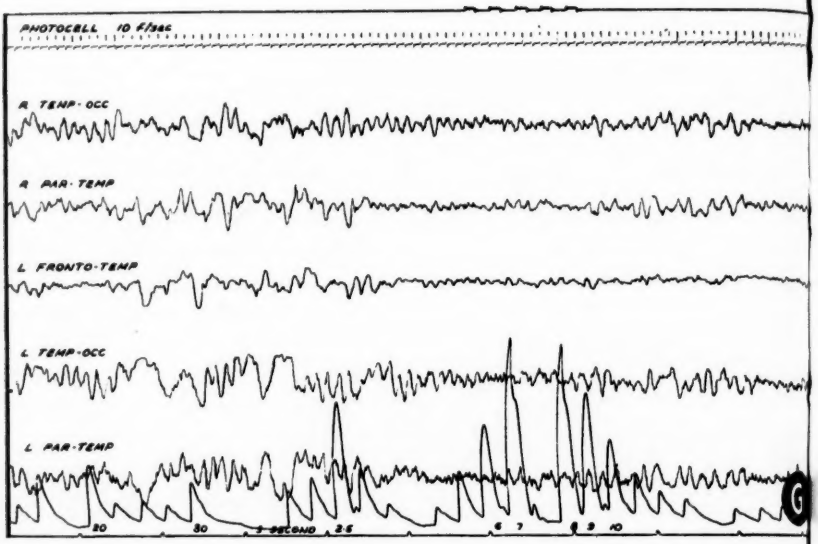


Fig. G. *Abnormal Flicker Response.* Same subject as in *Fig.* F. High voltage slow disturbances are mented during flicker at 10 f./sec. These are far greater and slower on the left than the right. No that the dominant delta component, 2.5, is the 4th subharmonic of the stimulus frequency—3rd and low subharmonics are often prominent in unstable EEG flicker responses.[26, 27]

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The Significance of Manganese in Human Tissues

II: The Presence of Manganese in Human Tissues

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The presence of manganese in human tissues invites attention from several points of view. On account of its general occurrence in nature, in rocks and soils, manganese is widely distributed in plants and it may therefore confidently be expected to occur normally in human tissues. Manganese is an essential element for plant development being located mainly in the leaves and actively growing parts of plants. Various authors have indicated its possible importance as a necessary factor in animal metabolism and, although opinions are generally guarded, it seems to be accepted that manganese is an essential element for human development.

It has been found that, in tissues, manganese is associated with the enzyme arginase, in the hydrolysis of arginine to urea and ornithine.[7] Other activities have been attributed to manganese such as the stimulation of tissue respiration and activation of other enzymes such as peroxidases. Its presence in human tissues may, therefore, be of importance from a nutritional point of view, and the amounts of manganese in various tissues, together with a comparison of these amounts between different races, should be of interest in this respect. From a toxicological point of view, the salts of manganese, with the exception of the permanganates, produce no noticeable effects. Chronic manganese poisoning, resulting from exposure to the dust, particularly of the oxides produced in grinding processes, does, however, form an industrial hazard. Although poisoning by permanganate is not common, it is essential in such cases to know what the normal manganese content of human tissues is so that definite conclusions may be drawn regarding the possibility of poisoning.

Considerable work has been carried out on the manganese content of human and animal tissues and Monier-Williams[7] has summarized much of this work. The manganese content of human and animal tissues and organs ranges from 0.2 to 4.0 parts per million, the largest amounts being found in the liver and pancreas. McCrae[6] has found 1.5–1.6 parts per million of manganese in hair. There is no clear evidence that a manganese deficiency can occur in human beings but Everson and Daniels[3] suggest that children of 3 to 5 years should receive daily 0.3–0.5 mg. per kilogram of body weight. Basu and Malaker[1] give 4.6 mg. daily as the amount required to keep an adult male in manganese balance. It was felt that data on the

manganese content of the tissues of human beings in South Africa might be of interest, particularly as a comparison could be made between the data for the two main races in this country.

In the present work, opportunity was taken of the fact that many viscera of the different races are examined in this laboratory in cases of poisoning. Those cases, in which arsenical poisoning was suspected were used, since the preparation of the final solution required for the determination of arsenic provided a solution which could also be used for the determination of manganese. The actual determination was based upon the oxidation of the manganese to permanganate by means of periodate and has been described elsewhere. (This *Journal*, p. 55.)

The detailed results for the manganese content of the liver, the kidneys and the stomach of Europeans, Natives and Native children (up to the age of 12 years) have been condensed and the data are reported in Table I in terms of mg. per kg. of organ.

Table I

Race	*Organ*	*Number*	*Average*	*Maximum*	*Minimum*	*Stand. Deviation*	*Coefficient of Variation*
European	Liver	10	1·33	1·60	0·83	0·250	18·9
	Kidneys	9	0·76	1·05	0·50	0·220	29·0
	Stomach	9	0·49	0·83	0·28	0·172	34·9
	Intestines	8	0·64	2·04	0·35	—	—
Native	Liver	17	1·46	2·09	1·05	0·353	34·1
	Kidneys	13	0·75	1·00	0·57	0·134	17·8
	Stomach	13	0·70	1·27	0·22	0·305	43·9
	Intestines	6	0·81	2·02	0·39	—	—
Native Children	Liver	11	1·67	2·37	1·10	0·488	29·1
	Kidneys	10	1·15	1·87	0·74	0·344	30·0
	Stomach	8	1·45	4·07	0·50	1·570	108·3
	Intestines	3	1·13	1·23	1·05	—	—

It will be noted that the manganese contents of European and Native livers and kidneys are very similar, the differences being 0.13 mg. and 0.01 mg. respectively. These differences are not significant when tested by the 't' test of Fisher,[4] the values being 1.09 and 0.10 while the values for a 20 to 1 significance should be 2.06 and 2.09. These results indicate that no conclusions can be drawn concerning the effect of race upon the manganese content of these organs. The results for the stomach are, of course, subject to temporary factors and, as shown by the coefficients of variation, are liable to wide fluctuations. Thus they do not form a satisfactory basis for comparison. Similar conclusions apply to the results for the intestines.

It is of interest to note that, in the case of the liver, there is a wider scatter of the values in the results for Natives and it is pertinent to enquire whether this may not be due to dietary factors. The results were therefore grouped into the two classes of rural and urban Natives. It was found that

the average values were 1.56 and 1.16 respectively. Owing to the small number (4) of urban results available it was not possible to test rigidly the significance of this difference but the value of 't' was found to be 3.0 The value for a 20 to 1 significance should be 2.13. This result would seem to indicate that dietary factors play a part in influencing the manganese content of livers. The urban Native is naturally more prone to indulge in the more varied European type of diet.

The results for Native children are of interest. Although the manganese content of the livers of Native children are somewhat higher than those for either the European or the Natives, the differences are not highly significant, the values of 't' being 1.98 and 1.27 respectively. The value of the manganese content of the kidneys of Native children, however, was significantly higher than that of the European or the Native kidneys. The differences were 0.39 mg. and 0.40 mg. respectively, the value of 't' for a 20 to 1 significance being 2.84 and 3.78 with the 20 to 1 significance limit being about 2.08. These differences would appear to be due to a physiological factor associated with a greater ease of absorption through the walls of the alimentary tract in the case of children.

In order to obtain an idea of the absolute amount of manganese in the organs, the weight of manganese per organ has been given in a condensed form in Table II in terms of mg. per organ.

Table II

Race	*Organ*	*Number*	*Average*	*Maximum*	*Minimum*	*Stand. Deviation*	*Coefficient of Variation*
European	Liver	10	1·93	2·43	0·58	0·518	26·8
	Kidneys	9	0·20	0·32	0·11	0·065	32·7
	Stomach	9	0·15	0·30	0·07	0·038	59·5
Native	Liver	15	1·42	2·00	0·85	0·321	32·6
	Kidneys	13	0·16	0·36	0·07	0·071	43·5
	Stomach	13	0·17	0·36	0·05	0·098	56·8
Native Child	Liver	11	0·62	1·00	0·24	0·257	41·3
	Kidneys	10	0·08	0·16	0·02	0·046	56·5
	Stomach	8	0·17	0·53	0·03	0·211	123·4

It will be noted that the amount of manganese in both the liver and kidneys is greater in the European than in the Native. The average differences were 0.51 mg. and 0.04 mg. respectively. The 't' values for the differences were 3.14 and 2.44, the value of 't' for a 20 to 1 significance being about 2.08. A closer examination of the data, however, indicated that the average weight of the European liver was greater than that of the Native. The average weights of the livers used in the present work were found to be 1430 gm. and 990 gm. for the European and Native respectively. The 't' value for the difference was 4.36 which was highly significant. In order to confirm this result a further series of weights of 35 Native livers and

25 European livers were selected at random. The average weights were found to be 1695 gm. and 1164 gm. respectively and the 't' value was 6.95. In the case of the kidneys the average weights in the present work were 257 gm. and 213 gm. for Europeans and Natives respectively and the 't' value of the difference was 1.84, which has a significance of about 15 to 1. With the larger range of weights, mentioned above, the average weights of the kidneys were found to be 294 and 242 gm. The 't' value for the difference was 2.88 which indicates a greater average weight for European kidneys. These facts would therefore account for the significantly greater weight of manganese in European livers and kidneys than in Native livers and kidneys.

For similar reasons, viz. the relatively small weights of the organs, the amounts of manganese found in the organs of Native children were distinctly less than in the corresponding organs of adults.

In Table III the mean of two sets of results for the manganese content of the same organs of a mixed race are reported.

Table III

	Mg. per Kg.	*Mg. per Organ*
Liver ..	1·61	1·01
Kidneys ..	1·02	0·12
Stomach ..	0·33	0·04

Table IV

Organ	*Manganese*	*Average*
Blood	0·21, 0·11, 0·17	0·16
Brain	0·28	0·28
Heart	0·55, 0·20	0·38
Hair	1·9, 1·4	1·65
Finger Nails ..	6·7, 6·8, 4·6	6·03
Toe Nails	4·1	4·1
Stomach Contents ..	0·39, 0·16, 1·04	0·53
Urine	0·06	0·06
Faeces	3·5, 3·3	3·4

These results are of the same order of magnitude as those which have been discussed but no comparisons can be drawn owing to the insufficiency of the data. In view of the results given in Tables I and II, it would not appear that any significant differences could be expected between the different races.

It will be noted that manganese occurs to a greater extent in the liver than in the kidneys. The distribution of manganese between the liver and kidneys seems to be the same in the cases of the European and the Native, the average ratios being 1.91 and 1.96 respectively. In the case of the Native child there appears to be relatively somewhat less manganese in the kidneys than in the liver, the ratio being 1.52. However, the results seem to be in accord with the usual type of distribution where the liver stores a greater proportion of the element than does the kidney. The distribution of manganese follows in this respect the distribution of lead.[2]

To obtain some indication of the distribution of manganese in the human system, the manganese content of a few other tissues have been determined when samples have been available. The results are given in Table IV in terms of mg. per kg.

These results are of interest from the point of view of the absorption and excretion of manganese in the human system. The high content in the nails as compared with that in the hair is of interest and, in this respect, resembles the distribution of arsenic. The result for faeces in comparison with that for the urine seems to suggest that manganese is eliminated chiefly through the intestinal tract. Kent and McCance [5] found that even on high manganese diets the urinary excretion was less than 0.1 mg. The data for blood, brain and heart indicate that the absorption by these organs is limited.

In order to compare these results with those for animal organs, opportunity was taken in 3 cases to determine the manganese content of certain tissues from 3 cows. The results are given in Table V in terms of mg. per kg.

Table V

Organ	*Average*	*Maximum*	*Minimum*
Liver	3·60	5·22	1·73
Kidney	1·12	1·43	0·72
Stomach	10·12	17·05	2·31
Stomach Content ..	20·35 (one)	—	—

These results are interesting in view of the relatively high levels of manganese found in the tissues. Those for the stomach contents are particularly interesting. These results may be ascribed to the fact that this animal is herbivorous. It is well established that the manganese content is higher in plants than in animals and, in addition, there is no doubt that considerable quantities of adherent soil must be ingested with the plant material, which forms the major item of their diet. It is of interest that the average ratio between the manganese content of the liver and that of the kidneys in this case is 3.08, compared with 1.91–1.96 in the case of human beings. This distribution might be ascribed mainly to the difference in diet.

The author would like to thank those members of the Staff of the Government Chemical Laboratories who supplied the required solutions for this work.

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REVIEWS OF BOOKS

Doctors in Court

The Roentgenologist in Court. By Samuel Wright Donaldson, M.D., F.A.C.R. (Pp. 348 + xiv. 2nd ed. 55s. 6d.) England: Blackwell Scientific Publications Ltd., 1954.

Contents: 1. Preface. 2. An Appreciation. 3. Introduction. 4. The Relationship Between Physician and Patient. 5. Malpractice. 6. The Physician and the Law of Agency. 7. Malpractice, Defence and Prophylaxis. 8. Evidence and Testimony. 9. Privileged Communications. 10. Expert Testimony. 11. Expert Witness Fees. 12. X-ray Films as Evidence. 13. Ownership of Films. 14. Physicians and Contracts. 15. "Doctor, Take the Stand." 16. X-rays and Dentistry. 17. Odds and Ends. 18. Conclusion. 19. Bibliography. 20. Key to Citations. 21. Table of Cases (Alphabetical). 22. Table of Cases (by States). 23. Index.

Though primarily addressed to roentgenologists, this book has little in it that does not have an equally direct bearing on the activities of non-radiological practitioners. It may be particularly pertinent for radiologists to know that blondes are more sensitive to X-rays than brunettes (p. 92); but all doctors are equally interested in how to give evidence in court and to prepare for cross-examination; the ethical and legal obligations imposed by the secret doctor-patient relationship; in whom ownership of records vests, whether these be X-ray films, ECG tracings, photographs, etc.; the precautions to be taken when publication of recognizable illustrations of patients is contemplated; the legal implications of partnerships, restraint clauses, employment of an assistant or a locum tenens and other such contractual problems.

The increase in malpractice actions against doctors is an alarming feature of our times. In America they grow yearly. 'Four thousand physicians were sued for malpractice in 1937, and each and every one of those suits conditioned the community for a subsequent suit.' (Page 290). As Ruggeri has recently pointed out (*Rockey Mountain Medical Journal*, 1954, **51**, p. 880): 'The misunderstanding and the ill will engendered by the great number of malpractice actions has tended to break down the confidence of the public in the physician. It has been estimated that between 50 and 80 per cent of all actions for malpractice would be eliminated if thoughtless and inadvertent criticism of one physician of the work of his fellow physician could be stopped.'

Dr. Donaldson shares this view and gives excellent advice (based on an experienced study of the problem) about how to cope with this ever-present danger in clinical practice.

The author deals also thoroughly with that uniquely American phenomenon—the professional medical witness, and the special situation which affects expert witness fees in the U.S.A. This latter survey should serve as a practical and valuable guide to the perplexed physician about his rights as well as his duties; it will certainly assist in protecting him against unscrupulous lawyers.

When must a practitioner breach his confidential relationship with his patient? Illuminating on this score is the fate of the physician who treated the late John Dillinger but failed to report his action to the authorities. He was sent to prison for 'conspiring to harbour and conceal a fugitive from justice'. This had earlier also been the fate of the Dr. Samuel Mudd who treated Lincoln's assassin—John Wilkes Booth.

There can be few medical practitioners who have not been the target of the 'hypothetical question' so often conjured up by counsel. This form of examination receives a thorough review, in the course of which the interesting point is made that the expert witness may not, in his reply, invade the province of the jury, e.g. in a malpractice case 'he cannot in his opinion state whether or not there was negligence' (p. 156). That decision is for the jury or the court and this sound principle possibly renders it less embarrassing for a physician involved in such an action on behalf of the plaintiff patient.

The text is profusely documented with cases which are arranged alphabetically as well as by States in a Table at the end of the volume. A comprehensive

index concludes a valuable account, entertaining as well as instructive, which holds much material deserving careful consideration by the established practitioner as well as his more junior colleague. At the same time, Dr. Donaldson's book clearly proves the need for more adequate instruction in these medical problems while the undergraduate is still at his Medical School.

Industrial Medicine and Hygiene

Industrial Medicine and Hygiene: Volume I. Edited by E. R. A. Merewether, C.B.E., O.St.J., M.D., F.R.C.P., D.I.H., F.R.S.Ed., Barrister-at-Law, H.M. Senior Medical Inspector of Factories, Ministry of Labour and National Service; Chief Medical Adviser, Ministry of Agriculture and Fisheries. (Pp. 405.) Butterworth & Co. (Publishers) Ltd., London, 1954.

Contents: 1. Work of the Factory Inspectorate. 2. Factory Legislation. 3. Statistical and Epidemiological Methods in Occupational Medicine. 4. The Organization and Operation of an Industrial Medical Department. 5. The Organization of Industrial and Occupational Nursing Services. 6. The Ministry of Supply Medical Service. 7. Medical Aspects of Coal Mining. 8. Medical Services in Inland Transport. 9. Medical Supervision in Shops, Warehouses and Offices. 10. The Young Worker. 11. The Disabled Worker. 12. Industrial Rehabilitation. 13. National Insurance Benefit for Sick and Injured. 14. Feeding the Worker. 15. Canteen Services in Industry.

The House of Butterworth has in the past published distinguished encyclopaedic works both in the field of law and of medicine. This tradition has again been observed in Dr. Merewether's undertaking, for which he is eminently qualified as he is a Barrister-at-Law who is also H.M. Senior Medical Inspector of Factories and Chief Medical Adviser to the Ministry of Agriculture and Fisheries.

The cautious way in which statistical data must be used is admirably illustrated in the Chapter on *Statistical and Epidemiological Methods in Occupational Medicine*, where Dr. Reid emphasizes that the 'recurrent difficulty in the interpretation of death rates by occupation is the disentangling of occupational from social factors . . . It is difficult to know whether the adverse mortality experience of the unskilled labourer is due to the circumstances of his work or to the bad diet, inadequate housing, and other social correlates of his economic status . . . If both husband and wife have an adverse mortality experience, then we can surmise that this excessive death rate is due to the unhealthy conditions of the home life which they share. If, on the other hand, an unduly high death rate among such husbands is not shared by their wives, we must deduce that the added hazard to life stems from a risk specific to their occupation and not from their social environment. The wives of physicians and surgeons, for example, have the low mortality rates which accord with their favourable social environment; but their unfortunate husbands have a higher-than-average death rate, which is presumably due to some added risk associated with their profession' (pp. 62-63).

It would be interesting to know whether lawyers and their wives share the hazards of their medical colleagues.

To advance from the study of groups to the treatment of the individual is also a difficult matter enjoining commonsense in the approach. Accident proneness and sickness proneness may exist in industry, but the practical value of the studies which led to these concepts has been disappointing, since so much depends on major environmental factors which seem to outweigh in importance the weeding out of the 'sickness and accident prone' (p. 83).

Dr. Fisher points out in the Chapter on *Medical Aspects of Coal Mining* that the current treatment of coal gas (carbon monoxide) poisoning by the administration of a mixture of 93% oxygen and 7% carbon monoxide, has been based on the high authority of Haldane. This view squared satisfactorily with the physiological teaching that carbon dioxide moves the oxyhaemoglobin dissociation curve to the right and makes it more sigmoid, thus improving the efficiency with which the blood gives up oxygen.

The hypothesis also explained the clinical collapse observed when victims were removed from a vitiated atmosphere to a purer one where there was plenty of oxygen, as the oxygen-dissociating power of carbon dioxide was thereby lost. It is therefore surprising to find that a recent (1952) report (p. 179) states that the value of carbon dioxide, even under medical supervision, is in doubt. Indeed the Committee concerned recommends that in *first aid* resuscitation only oxygen should be used, and it regards carbon dioxide as a brain depressant (anaesthetic) as well as a breathing stimulant, but one in which the stimulus to breathing diminishes as the anaesthetic effect increases. Consequently, it is argued, the more critical the situation, the more dangerous the carbon dioxide becomes.

The medical requirements, particularly for eyesight and hearing, which must be met in public transport drivers, provide a useful background for a discussion of

the medical tests which should be passed by private drivers, in whom the demands do not need to be so severe. This Chapter has an important bearing on road safety and, in the same way as the book as a whole, will find ready application in all parts of the industrialized world, whether factory and social legislation coincides with the British pattern or not.

Dr. Pyke has set out admirably the principles of nutrition in the Chapter on *Feeding the Worker*. Food is more than a matter of calculated calories. 'The choice of a dietary pattern appropriate to the type of job which a worker is doing always affects his or her contentment and well-being. It may also be important for the maintenance of health, and will certainly affect the efficiency with which the work is done' (p. 349). Moreover, the special problems involved in the change over to night-shift work may produce complaints about food unconsciously caused by a physiological unsuitability for night work, since Kleitman's interesting experiments have demonstrated that there are two distinct classes of persons as far as this reversal of the living pattern is concerned, viz. those who can and those who cannot quickly acclimatize themselves to the new conditions.

The second volume of what will become a standard work of reference for a rapidly growing field of activity has already appeared, and will be reviewed in the next issue of this *Journal*.

Craig and Bentley: Symptoms of a Pandemic?

Trial of Christopher Craig and Derek William Bentley (Notable British Trials Series). Edited by H. Montgomery Hyde, M.P. (pp. 264, Illustrated 15s). London, Edinburgh and Glasgow: William Hodge and Company Limited. 1954.

Contents: Introduction. 1. The Trial—First Day, 37; Second Day, 112; Third Day, 195. *Appendices:* I. Proceedings in the Court of Criminal Appeal, 210. II. Debate in the House of Commons, 27 January 1953, 227. III. The Law of Murder (Extract from the Report of the Royal Commission on Capital Punishment (1949-1953), 244 IV. *Regina* v. *Niven Scott Craig and Cyril Burney*, 260. V. Citations, 263.

Christopher Craig was 16 and his friend Derek Bentley was 19 when Craig wantonly shot and killed a policeman during their joint attempt to break into a warehouse.

Both boys came from good families, and there seemed, on the face of it, no good reason for them to have embarked on a career of crime.

These two youngsters pose the problem (which may become catastrophic for Society) of the modern youthful criminal. Their demeanour before and during the trial is reminiscent of the New Zealand girls Pauline Parker (16) and Juliet Hulme (15) who murdered Pauline's mother and showed no concern, remorse or regret about what they had done.

Juvenile delinquency is not something new. As Mr. Montgomery Hyde points out in his *Introduction*, the world's first reformatory 'for the correction and instruction of wayward boys' was built in Rome at the beginning of the 18th century. But it is the magnitude of the problem, especially since World War II, that has created understandable alarm all over the world.

In Britain youthful crime has more than doubled since the last War, and crimes of violence (including murder, rape, robbery, etc.) have almost quadrupled, over half the delinquents being between 17-21. Mr. Hyde points out that the 'increase has been particularly marked among the youths who have grown up under Britain's Welfare State' (p. 1), but he does not discuss whether this is *post hoc* or *propter hoc*.

Rebellious violence without remorse, conscience or apparent good cause is characteristic of the young psychopaths of our time. Dr. Robert Lindner (the well-known American authority in this field) postulates that the fantasies, adolescent ambitions and frustrations have heretofore been 'contained within the skin's envelope, merely felt and suffered in the private agony of a tormenting preadulthood'. The storm was kept inward, and often sublimated into creative and fructifying activity, whether in the domain of the arts or the sciences. But to-day the inward eye of the adolescent does not seem to flash so readily with a poetic inspiration; and creative solitude is abandoned easily in favour of pack-hunting in gangs and herd activity in which the individual conscience is drowned. Craig and Bentley typify a social disease which is virtually pandemic and their state of chronic mutiny marks a climax in the history of delinquency in the United Kingdom.

Public clamour was aroused when it was learned that Bentley was to be executed, despite the jury's recommendation to mercy. (Craig was automatically spared because of his age).

An interesting legal point arose concerning how long common responsibility endures. Mr. R. T. Paget, Q.C. has argued that it endures only so long as the common design endures. Because Bentley was arrested on the roof-top of

the warehouse at an early stage on that fateful occasion, had he not 'withdrawn from the common resistance and determined to go quietly' (p. 28), thus excluding himself from further culpability? This was not put to the jury in the trial and, in any event, Bentley's appeal was dismissed by the Court of Criminal Appeal.

If age is to be a ground for compassion, Bentley's mental quality raises another interesting problem. Still a youth in his teens, he was undoubtedly dominated by the forceful and aggressive personality of the younger boy. He was considerably retarded in intelligence, and was well below the average. His mental age (or I.Q.) would have provided a sounder basis for a just evaluation of his responsibility, and a truer assessment of the importance of his physical age. Even if this could not have swayed the judicial mind in court, it might have provided proper grounds for clemency once the matter reached the Home Secretary.

Bentley appears to have been the first individual in English legal history to have been 'hanged as an accomplice in a crime for which the principal in the first degree could not be executed on grounds of age' (p. 31). The trial therefore justifies classification as *Notable*, and the Appendices covering the proceedings in the Court of Criminal Appeal, the debate in the House of Commons and the extracts from the Royal Commission on Capital Punishment (1949-1953) make this verbatim record a very valuable addition to the forensic library.

Progress in Forensic Medicine

The British Encyclopaedia of Medical Practice, including Medicine, Surgery, Obstetrics, Gynaecology and Other Special Subjects. Medical Progress, 1954. Editor in Chief: The Lord Horder, G.C.V.O., M.D., F.R.C.P. Extra Physician to H.M. The Queen, Consulting Physician to St. Bartholomew's Hospital, London. (Pp. 281 + Index.) London: Butterworth and Co. (Publishers) Ltd., 1954.

Contents: Part I. Critical Surveys: 1. Medicine. 2. Surgery. 3. Obstetrics and Gynaecology. 4. Chest Surgery. 5. Neurology. 6. Gastro-Enterology. 7. Psychological Medicine. 8. Occupational Skin Diseases. 9. Progress in Forensic Medicine. 10. Physical Medicine. 11. School and Priority Dental Services.
Part II. Drugs: 12. Recent Developments in Pharmacology and Therapeutics.
Part III. Abstracts: 13. Abdominal Emergencies—Whooping Cough.
Index.

For readers of this *Journal* the main interest will centre in Dr. Keith Simpson's review of *Progress in Forensic Medicine*, which highlights many recent advances very succinctly. Dr. Simpson deals with the following:

Estimation of the early post-mortem interval; Fluidity of the blood after death; Exhumation: Problems of identity (age, sex and stature); Blood groups; Neonatal problems (Separate existence, death—including unexpected sudden death in infancy); Obscure sudden death in adults, including obscure poisoning; Asphyxia, including the significance of the so-called asphyxial petechiae; Brain injury, including the EEG and the M'Naghten rules; Alcohol and traffic accidents; Changes in recent medical law in the United Kingdom.

Considerably greater attention is being paid in many parts of the world to the increasingly helpful role of the laboratory in connection with alcohol estimations in the blood. Norway and Sweden fix arbitrary blood levels of 50 and 80 mg. per 100 c.c. to establish culpability, and 150 mg. per 100 c.c. for a mandatory prison sentence, although it is generally recognized that only about 50% of people are intoxicated at this concentration. In Denmark the biochemical evidence is considered together with the clinical evidence—a course which has much to recommend it.

Dr. Simpson reports that the 'drunkometer' is losing support, even in the United States. Certainly breath alcohol levels have not found much support elsewhere.

Practical guidance for cross-examination is provided in a list of 10 points (pp. 111-112). These can be added to by pointing out that the factor for the alcohol in the blood (labelled 'r' by Widmark) is not fixed at 0.7, but has a considerable range. Alha (1951, Ann. Acad. Sci. Fenn, Series A., v., Medica-Anthropologica, 26) has established that the 95% confidence limits of the individual observations were 0.58 and 0.87.

It should be appreciated that statistical means are of scant assistance in the individual case, and the interests of justice are best served by recognizing the considerable variation that may exist normally.

In the same way, Alha found the rate of alcohol elimination to vary from 55.3 to 140.5 mg. per kg. body weight per hour. Dr. Simpson's figures of 7 gm. per hour in the male and 5 gm. per hour in the female should be modified in the light of Alha's important work, which could appropriately be incorporated in the next account of *Progress in Forensic Medicine*.

The review of sudden death in in-

fancy should do much to dissipate that ready tendency to diagnose cot asphyxia or overlaying, inherited from a less critical generation of forensic pathologists. Fully warranted, also, is the emphasis on carbon monoxide poisoning, so likely to be overlooked as a cause of death in the elderly. This Dr. Simpson himself has ably demonstrated.

The gradual transition of the human foetal lung from a glandular to an alveolar form has been wrongly attributed to Barcroft; and Keen's important observations on the estimation of stature from the long bones have been overlooked (J. Forensic Med., 1953, 1, 46.)

Dr. Simpson's review appears for the first time in this *Progress* supplement to the *Encyclopaedia*. It is timely current comment, and as an occasional, perhaps annual, survey it will provide most valuable interim reports.

Treatment of Alcoholics

The Treatment of the Alcoholic. By Fritz Kant, M.D. (Pp. 130—vi. 25s.) England: Blackwell Scientific Publications, Ltd., Oxford. 1954.

Contents: 1. Introduction. 2. Why do People Drink? 3. Signs Common to Alcohol Intoxication. 4. Extent of Problem and Attitudes Toward Drinking. 5. The Alcohol Addict. 6. How to Detect the Alcoholic. 7. Abnormal Personality Types Among Alcoholics. 8. Causes of Alcoholism. 9. Who Can be Treated. 10. The Addict and His Family. 11. Paranoid Trends Toward the Wife. 12. Alcoholism and Neurosis. 13. Bringing the Addict for Treatment. 14. Mental Reactions and Physical Defects in Chronic Alcoholism. 15. Medical Treatment of the Alcoholic. 16. Psychotherapy. 17. Hospital or Outpatient Treatment. 18. Adjuncts to Psychotherapy: (*a*) The Conditioned Reflex Treatment; (*b*) Antabuse. 19. Alcoholism and Drug Addicts. 20. Alcoholism and Drug Addiction. 21. The Twelve Steps of A.A. 22. Integrated Therapy. 23. Prevention. 24. The Outlook. 25. Glossary. 26. Selected Bibliography. 27. Index.

Professor Kant's book, directed to physicians and judges, ministers, social workers and many others concerned with helping the alcoholic in our midst, provides 'a simple way to obtain the factual information needed'. He concludes that alcoholism is the manifestation of a neurosis, based on a great complexity of underlying causes, and that, like the poor, we will always have it with us.

Although complete eradication cannot be accomplished as has, e.g. been done for some bacterial diseases, there is no ground for defeatism or despair. 'The realization that alcoholism will develop in a certain number of people is not a reason for pessimism. It emphasizes the need to give the treatment program for alcoholism an accepted and permanent place in our society. The last two decades have clearly demonstrated that the alcoholic can be helped' (p. 114).

Alcoholic addicts drink to escape from a situation that weighs them down or creates undue tensions. This escapism enters to some extent even into normal social drinking, so that for many years the abnormal drinker is masked as merely a heavy social drinker, the serious maladjustment underlying his intemperance escaping recognition for a time.

Professor Kant does not share the view that periodic compulsive drinking (dipsomania) is an established entity, but he endorses the view that so-called symptomatic alcoholics may be an expression of mental illness, e.g. schizophrenia, manic-depressive psychosis, general paresis and certain epileptics. Indeed, excessive drinking may be the first sign of a beginning psychosis.

The fundamental cause of alcoholism is often postulated by diametrically opposed hypotheses. Latent homosexualism (as in *The Lost Weekend)* is endorsed by some psychiatrists, whereas Smith places it on a physiological (metabolic) basis, due to the pituitary gland.

This endocrine approach is of significance for the hormone treatment which relies on ACTH in controlling delirium tremens. Williams, on the other hand, blames a nutritional deficiency. His theory depends on experimental work with rats. The multiplicity of theories illustrates what is really a multiplicity of causes.

If the alcoholic is a neurotic, he is a neurotic with a difference. Psychotherapy alone will never be adequate, because he is an addict, who has formed a habit which provides pleasure and escape, and he is vulnerable to insidious social pressure in a society which 'glorifies wine and song in prose and verse'.

Professor Kant makes us face up to the social magnitude of the problem and provides a balanced survey of treatment and prevention. The latter is one of the most important ways of dealing with the matter.

The absence of sentimentalizing makes for a reliable, factual and convincing account which should attain its objective.

Police Training

Daily Training Bulletin of the Los Angeles Police Department. By W. H. Parker (Chief of Police). (Pp. 255—illustrated. 54s.) Oxford: Blackwell Scientific Publications.

Contents: 1. Personal Aids. 2. Important Public Relations Services. 3. Appearance in Court. 4. Police Files. 5. Sickness or Injury Reporting. 6. Lie Detector Tests and Preliminary Investigation. 7. Evidence. 8. The Use and Care of Firearms. 9. Communications Equipment. 10. Police Automotive Equipment. 11. Vehicle Impounds. 12. Field Tactics—Preliminary Investigation. 13. Arrest. 14. Traffic Violations. 15. Intoxication. 16. The Mentally Ill. 17. Civil Disturbance Control. 18. Major Disaster Control

The *Daily Training Bulletin of the Los Angeles Police Department* grew from a roll call training tradition which in its turn started spontaneously when some of the supervisors in the patrol divisions 'felt an obligation to pass on their experiences to the policemen under their supervision'. Although the law involved is necessarily that of California, Los Angeles and the regulations of the Department concerned, the scope of the instruction overrides the parochial limits set by geography and provides a manual which law enforcement officers all over the civilized world can and should study with profit to themselves and the public they are appointed to protect.

The Chapter on *Lie Detector Tests* is commendably brief, the point being made clearly that there is no machine capable of detecting lies, but that *key* questions may record *symptoms* of lying. The difficulty, of course, is that these symptoms are not characteristic of lying and can be due to emotional stress which has an innocent origin. Particularly objectionable, also, is the possible use of the instrument as a 'third degree' method of inquiry. In certain circumstances the machine can assist preliminary investigation, and this limited function is recognized very clearly.

Interestingly enough, the Atomic Energy Commission recently abandoned the lie detector as a tool in its security programme.

The Chapter on *Intoxication* sets out fully not only the procedures police officers must follow when dealing with persons charged with being under the influence of intoxicating liquor, but also includes a comprehensive set of clinical tests on the accused person's balance, gait, co-ordination (of fine movements), speech and reaction of the pupils to light. These tests are apparently proper in California when administered by lay persons; in most Western countries they would certainly be regarded as falling within the province of the medical expert.

The important warning is sounded that certain mouth washes leave an alcoholic odour for 10-15 minutes, so that an officer should not be 'misled into concluding that a driver has been drinking alcoholic liquor merely because his breath has an alcoholic odor' (p. 223).

Of forensic interest also is the handling of the mentally ill (Chapter 16) and the section dealing with how to give evidence in court would repay study by all witnesses, whether on or off the force. The graphic and entertaining illustrations in which every page abounds are one of the most helpful and striking features of this training manual, which makes its points tersely in words as well as in pictures.

A set of review questions at the end give an excellent opportunity to the reader to test himself on whether he has learned his lessons. Certainly the book should be employed extensively in training in police procedure, whether Californian local law is applicable or not.